THE

JOURNAL OF

COMPARATIVE PATHOLOGY

AND

THERAPEUTICS

(With which is incorporated The Veterinarian)

EDITED BY

J. M'FADYEAN, M.B., B.Sc., M.R.C.V.S.
ROYAL VETERINARY COLLEGE, LONDON

AND

JOHN A. W. DOLLAR, M.R.C.V.S., F.R.S.E.
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AN OUTBREAK OF DIPHTHERIA ASSOCIATED WITH A SIMILAR DISEASE AMONG FOWLS AND A VESICULAR ERUPTION ON THE UDDERS OF COWS.

By Lieut. J. D. E. HOLMES, B.A., M.R.C.V.S., Indian Civil
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THE RELATION OF MILK SUPPLY TO HUMAN DIPHTHERIA.

MANY years before the discovery of the specific organism of human diphtheria, it was recognised that there existed in many epidemics some connection between the disease and the milk supply of the affected area. Also, it had been frequently observed, especially on the Continent, that cases of human diphtheria had been preceded by an epidemic of a similar disease among fowls. The discovery of the Klebs-Löffler bacillus as the causal agent of diphtheria has not, up to the present time, made clear what rôle, if any, is played by the cow and fowl in the dissemination of the disease. The theory that milk is capable of acting as a vehicle of infection is one that is generally accepted. There is undoubted evidence that in certain epidemics the milk supply was the means by which the infection was conveyed. In some instances it was suspected that the milk had become contaminated by human agency, but in other cases no extraneous source of infection was discoverable, and it was believed that the infective material had come from the cow herself.

The Annual Reports of the Medical Officers to the Local Government Board furnish many interesting facts bearing on this question. In the Report for the years 1889-90, an epidemic in Macclesfield

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district is described. It was noticed that almost all the patients had procured their milk from the same dairy, and that those members of each household who were the chief drinkers of raw milk were attacked first. On enquiry at the dairy it was ascertained that some days previous to the outbreak the milk of a newly purchased cow, which had recently calved, had been added to the stock of milk. This cow was segregated and the issue of her milk stopped, and from that time no fresh cases of diphtheria occurred. After some weeks the milk of this cow was again sold, and soon afterwards fresh cases of diphtheria followed among the customers who had used it. On the teats and udder of this cow vesicles and pimples were found at the time of the second series of cases.

Dean and Todd[1] investigated an outbreak which arose from a diseased condition of the udders of the cows from which the family obtained their milk. They found virulent diphtheria bacilli in certain ulcers on the teats and udders of these cows.

Bowhill[2] isolated a virulent diphtheria bacillus from suspected milk in connection with an outbreak at Cardiff.

In other instances organisms morphologically resembling the Klebs-Löffler bacillus have been demonstrated in milk, but they proved to be non-virulent when tested on guinea-pigs.

Klein[3] inoculated milch cows subcutaneously with cultures of diphtheria. A tumour appeared at the seat of inoculation and subsided in a few days. On the teats and udder three to four days after inoculation vesicles, pustules, and ulcers appeared. In the lymph from these induced vesicles the diphtheria bacillus was demonstrated both in cover-glass specimens and by cultures. These cows showed a slight febrile reaction during the first two or three days, and coughed slightly while the eruption manifested itself. Cultures from the milk showed diphtheria bacilli. Eventually the cows succumbed to the disease, one after fourteen days, the other after twenty-four.

In 1890 Klein[4] observed ulcerative eruptions on the udders of cows whose milk was suspected to have conveyed diphtheria. The eruption was similar to that produced by experimental inoculation.

In brief, on such and similar evidence is based the theory that the cow is capable of becoming an active agent in the dissemination of the disease.

The facts do not carry us further than that it seems probable that the infective material of diphtheria can be harboured in the body of the cow and conveyed in her milk.

We have no knowledge as to how the cow, in the first instance, becomes infected. Some authorities assert that cows act solely as agents in transmitting the disease, that they take up the material of human diphtheria in their food and secrete it in their milk without being themselves in any way affected.

A more probable theory is that the cow takes up some organism, a facultative agent of human diphtheria, from the soil with her food, that this organism affects her to a slight extent, causing cough and eruptions on the teats and udder, and that by the passage through

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[1] "Journal of Hygiene," Vol. II., p. 194.
[2] "Veterinary Record" 8th April 1899.
[3] "Local Government Board Report," 1889.
[4] "Local Government Board Report," 1890

her body it acquires further powers and facility for acting on human beings. If this be the case, it would not necessarily follow that the organism which is the primary agent of the disease in the cow is morphologically identical with the Klebs-Löffler bacillus. There is perhaps no other known pathogenic organism which presents itself under so many varieties of form according to the conditions of its growth. Nor would it be necessary for this organism to display the same virulency for animals as that recovered from the human body.

Our methods for differentiating the diphtheria bacillus are still so very unsatisfactory, and our knowledge of its life cycle so imperfect, that we have to exclude from this species all organisms which are not identical in shape and equal in virulency for the guinea pig to the Klebs-Löffler bacillus.

THE RELATION OF AVIAN TO HUMAN DIPHTHERIA.

The question of the relation of fowl to human diphtheria is one which has for many years excited controversy among scientists, and which at the present time is far from being decided. There are many different forms of the disease in the common fowl and in other birds, and many different organisms have been described as the agent of avian diphtheria.

According to Nocard,[1] the bacillus of avian diphtheria is analogous in its form to that of the hæmorrhagic septicæmias. He considers that it may be classed among the pseudo-diphtheria bacilli, and that it is pathogenic to man.

Moore[2] described a Pasteurella identical with that of fowl cholera.

Löffler[3] states that diphtheria in pigeons is due to a non-motile bacillus similar to that of septicæmia of rabbits.

Many observers have described forms analogous to the bacillus of Klebs-Löffler. Gallez[4] found in the nasal mucous membrane of fowls affected with contagious coryza a bacillus identical in colour-reaction and culture with the Klebs-Löffler bacillus.

The same organism was observed by Ferre[5] in diphtheritic lesions among fowls, and Gratia and Lienaux[5] isolated a similar bacillus from the mouths of pigeons suffering from diphtheria.

Dr Bruno Galli Valerio,[6] in a summary on the question of the identity of diphtheria in man and the fowl, gives a number of interesting facts. He quotes a saying of Gallez on the subject : " If medicine, as formerly, had remained a science of observation, in presence of the relatively enormous number of facts brought to-day in support of this hypothesis, no one would dream of doubting it, and the identity of animal and human diphtheria would be an established fact."

Among others, Boing,[6] Hingworth,[6] Bilhaut,[6] and Esmmerlich have cited instances in their experiences of direct transmission of diphtheria from fowl to man. Chauveau[6] considered the diphtheria of man and birds identical. Numerous other observers have recorded cases of

[1] Nocard et Leclainche. "Les Maladies Microbiennes des Animaux." 3rd Edition 1903.
[2] "United States Bureau of Animal Industry Bulletin" No. 8. 1895.
[3] "Miltheil. des Kaiserl. Gesundheitsamtes" t. II., 1884, p. 421.
[4] "Annales de Médecine Vétérinaire." 1895, p. 309.
[5] Nocard et Leclainche. "Les Maladies Microbiennes des Animaux." 3rd Edition 1903.
[6] "Centralblatt für Bakteriologie" Vol. XXII., p. 500.

diphtheria among people having charge of, or living in close contact with,. fowls affected with the disease.

Guerin[1] is perhaps the latest worker on the question of the identity of the human and the avian disease. As the result of his experiments and of his observations of numerous epidemics of fowl diphtheria, he concludes that human diphtheria is entirely distinct from that of the fowl. The agent of the fowl disease is, he states, a Pasteurella. He found a difficulty in transmitting the disease from fowl to fowl, and in order to raise the virulency of the contagium he had to pass it three or four times through pigeons, by inoculating them in the conjunctiva with material from the false membrane of an affected fowl. The third or fourth pigeon died in twenty-four hours with all the symptoms of a general septicæmia. With this virus he inoculated subcutaneously other birds, which died in twenty-four hours, and the specific bacillus. was found in all the tissues. The disease is conveyed by ingestion and inoculation, but an intimate and long contact with the specific material is necessary.

Guerin in two instances isolated from the pseudo-membrane of an affected fowl an organism morphologically resembling the Klebs-Löffler bacillus, but it was non-virulent. He considers that these forms are identical with the pseudo-diphtheria bacillus frequently found in the throats of healthy human beings, and that they have no connection with true diphtheria.

THE MORPHOLOGY OF THE KLEBS-LÖFFLER BACILLUS AND ITS RELATION TO THE PSEUDO-DIPHTHERIA BACILLUS.

One of the most difficult problems in the study of diphtheria is the diversity of opinions as to what organisms are to be included among the specific agents of the disease.

The typical forms of the Klebs-Löffler bacillus, which is recognised as the specific agent of true diphtheria, are presented in a young culture on blood serum incubated at a temperature of 37° C. (Fig. 1.) The striking feature observed microscopically from such a culture is the variety of forms which are seen in every field. The peculiar arrangement of the bacilli, which lie generally at right angles or parallel to each other, is diagnostic. This has been compared with "spilled matches," or "a lot of pine needles on the ground."

Many of the forms contain two or more deeply stained granular bodies. Others show a central unstained space. Many other forms are typical, such as club, flask, comma-shapes, also coccus-like masses and bacilli with rounded ends and deeply stained granular bodies at either pole.

Westbrook, Wilson, and M'Daniel[2] have described nineteen different shapes. They recognise three main types ; (1) granular ; (2) barred ; (3) solid-coloured forms. It is probable that the diphtheria bacillus presents itself, according to the conditions of growth, under a greater variety of forms than has yet been recognised.

The shapes vary with the medium on which they are grown, with the

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[1] "Annales de l'Institut Pasteur" No. 12. 25th December 1901. and "Recueil de Médecine Vétérinaire" No. 1. 15th January 1903.
[2] "Trans. of the Assoc. of American Physicians," 1900.

age of the culture, and with the temperature to which it is submitted. (Figs. 1, 2, 3, 4, 5, and 10, *a*).

In older and degenerating cultures on agar there may remain nothing but a mass of coccus-like bodies. On examination under a high power, these are seen to be of oval shape, and in many a small central unstained space is observable. These, I found, if re-sown on blood-serum or on agar seldom reverted to very typical forms, and among them three shapes were constant. A small ovoid bacillus, some uniformly stained, some with a central unstained space; a rod-like bacillus of unequal breadth and varying length, uniformly stained; and a bacillus with rounded ends and unstained or slightly stained central space, and with deeply coloured granular bodies at either pole. These three forms were also constantly recovered from the tissues of animals which died after inoculation with the virus of human diphtheria, and also from the blood and tissues of animals dead from avian diphtheria. (Figs. 6, 7, 8, 9, 12, 14, and 15).

These when grown and subcultured on blood serum showed a greater variety of shapes, and presented greater resemblance to typical forms of the Klebs-Löffler bacillus, but they never regained the exact forms and arrangement of an original young blood-serum culture.

If a young and growing culture of the Klebs-Löffler bacillus be frequently and carefully examined under a high power, it will be seen that at first the granular bodies are small and not much more deeply stained than the other parts of the organism. After twenty-four to thirty hours their bodies become larger and more deeply stained, and the other parts take the stain faintly or not at all. Finally the granular bodies seem to burst forth from the body of the organism, and at this stage many free coccus-like forms may be seen in the field, with many bacilli faintly stained, some of which show one polar body remaining. This oval body is evidently a young form of the diphtheria bacillus, and it is probable that from it all the other various shapes have their origin, and that these forms are determined to a large extent by the conditions under which the oval body is developed.

The forms which we recognise as typical may be those developed by passage through human beings, whereas the simpler forms of ovoids, bi-polar stained bacilli, and rod-like uniformly stained bacilli may be more typical of a previous phase of existence.

In Fig. 12 swollen forms of the diphtheria bacillus are shown. Those in (*a*) were taken from an agar culture ten days old, transferred to blood-serum and then left at room temperature for twenty-four hours. A cover-glass specimen taken from the culture at the time showed these swollen forms. The culture was then incubated for twenty-four hours at 37° C., and on examination nothing but typical forms, as in Fig. 1, were observable.

Fig. 12 (*b*). This culture was taken from blood of a bull dead after inoculation with virus of human diphtheria. It was kept under like conditions, namely, on blood serum at room temperature for twenty-four hours. These swollen forms were then observed. After incubation for twenty-four hours their shapes disappeared, leaving typical forms.

Fig. 17 (*a*) shows somewhat similar swollen forms from blood of a rabbit dead after inoculation with virus of fowl diphtheria. The culture was kept and observed under like conditions.

The varieties in form of the diphtheria bacillus are so numerous and so imperfectly understood that we cannot decide the identity of a doubtful organism by the test of morphological similarity alone. Even regarding organisms which culturally and microscopically are identical with the acknowledged type of the Klebs-Löffler bacillus, there is much diversity of opinion as to what is and what is not to be classed among the species of true diphtheria bacilli.

Löffler[1] was the first to describe the presence in the false membrane of an organism closely resembling the diphtheria bacillus, but which was non-pathogenic for animals. Hofmann[2] and others studied the same organism. Löffler, Hofmann, and others consider it to be of an entirely distinct species, while Roux, Yersin,[3] and many other observers hold it to be an attenuated form of the true diphtheria bacillus. Cobbett[4] regards it as a different species, and believes that with practice it is possible to distinguish its forms from those of the Klebs-Löffler bacillus. Graham Smith[5] is of the same opinion.

The more general view is that it is morphologically identical with the diphtheria bacillus, and that the only point of distinction is that it is non-pathogenic for animals. Roux and Yersin,[6] starting with virulent cultures of the Klebs-Löffler bacillus, by growing them under unfavourable conditions in a current of air obtained a non-pathogenic bacillus. Hewlett and Knight[7] state that they have transformed the virulent into a non-virulent type, and *vice versa*. Richmond and Salter[8] record that they have succeeded in converting the pseudo into a virulent type by repeated passages through certain birds.

According to Sternberg[9] : " Recent authors are generally inclined to the opinion that bacilli which resemble the diphtheria bacillus in every respect except that they are non-pathogenic should be regarded as attenuated varieties of the diphtheria bacillus rather than as belonging to a distinct species—the so-called pseudo-diphtheria bacillus."

" The fact that the non-virulent type is very rare in fatal diphtheria and more common in benign cases, that it becomes more common in severe cases as they tend towards recovery, and that it is more observable in persons who have recently had diphtheria than in healthy individuals, renders the idea that the two microbes are entirely distinct difficult. The sharp distinction between virulent and non-virulent bacilli is arbitrary, and depends on the susceptibility of guinea-pigs."

THE DISTRIBUTION OF THE DIPHTHERIA BACILLUS IN THE BODY.

Formerly it was believed that diphtheria was a purely local disease, and that the specific agent was to be found in the false

[1] "Centralb. f. Bakteriologie," 1887.
[2] "Wiener Med. Wochenschrift," 1888.
[3] "Annales de l'Institut Pasteur," 1888-90.
[4] "Journal of Hygiene," Vol. I., 1901, p. 235.
[5] "Journal of Hygiene," Vol. III., 1903, p. 216.
[6] "Annales de l'Institut Pasteur," Vol. IV., 1890, p. 385.
[7] "Trans. Brit. Inst. Prev. Med.," 1st Series, 1897, p. 13.
[8] "Guy's Hospital Reports," Vol. LIII., 1898, p. 86.
[9] "Text Book of Bacteriology."

membrane alone. Wright,[1] Frosch,[2] Kartach, Stephens,[3] and others have demonstrated the Klebs-Löffler bacillus in the heart's blood, lungs, spleen, lymphatic glands, and kidney in fatal cases.

Abbott, Ghrisky[4] and Tarnik[5] have recorded that in exceptional instances diphtheria bacilli when deposited under the skin of the smaller animals may be found in the lymphatic apparatus of the omentum. Wright observed the diphtheria bacillus amongst inoculated animals in comparatively rare instances in liver, spleen, blood and kidney. Klein[6] found that when cows are inoculated subcutaneously diphtheritic lesions may appear in distant areas, and the bacilli may escape with the milk.

In the experiments recorded in this paper, among the animals inoculated with human diphtheria, and also among those inoculated with avian diphtheria, and which succumbed, in almost every instance the specific organism was observed in œdema, heart's blood, spleen, liver, and kidney. The forms of the human diphtheria bacilli varied somewhat in the different animals and in the various tissues, and were not very typical shapes. In blood-serum cultures they reverted to more typical forms, and when tested on animals they proved equally virulent as the original diphtheria culture.

HISTORY OF THE OUTBREAK.

Muktesar, at which the outbreak occurred, is situated on the Himalayas, at an altitude of about 7,600 feet. It is in a very isolated position, being twenty-three miles from the hill station of Naini Tal, and sixteen miles from Almora, a Ghurka cantonment. The nearest native villages are at a distance of three or four miles. Muktesar itself consists solely of the Bacteriological Laboratory of the Indian Civil Veterinary Department, with the residences of the staff and the necessary outbuildings for cattle and other animals. At the time of the outbreak there were two English families residing in the place. In one family there were three children between the ages of two and six, and in the other one child of about two years. These children had not been away from Muktesar, and had not met any other children for some months.

Diphtheria is a comparatively uncommon disease in India, and is generally met with in the plains and in a moist hot climate. As far as could be ascertained, there had not been any cases of diphtheria in Naini Tal, Almora, or in the surrounding villages for many years. No clue as to how or from where the infection could have been brought to Muktesar was discoverable.

About the second week in June (1903) one of the family of three children, a boy of two and a half years, was noticed to have a mucous discharge from both nostrils. Some days after the discharge commenced a few small ulcers appeared on the lip beneath the nose. The weather at this time was hot, and there was much dust in the air. The children were not eating well, and were somewhat out of sorts. Not much notice was taken of the discharge from the boy's

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[1] "Boston Med. and Sur. Journ.," Oct. 1794. [2] "Zeitsch f. Hygiene," 1893.
[3] "Journal of Bacteriology and Pathology," Vol. IV., p. 45.
[4] John Hopkin's Hosp. Bull., April 1893. [5] "Centralb. f. Bakteriol. u. Parasitenk," 1889.
[6] "Rep. Med. Off. Local Government Board," 1889, p. 167.

nose, as it was thought to be catarrh, due to a slight cold. He spoke as if he had a cold in the head, and sneezed frequently.

About the 5th of July the second child, a girl of four and a half years, complained of headache and appeared dull and listless, but showed no signs of any serious illness till the 12th inst. On that morning she again complained of headache, and lay in a chair all day. She sat up and ate some breakfast and lunch. During the day it was noticed that the glands on the right side of the neck were enlarged, and that the child had some difficulty in swallowing. The throat was examined, and a large patch of buff-coloured false membrane adherent to the back of the pharynx was discovered. This was touched with a small plug of cotton wool, and cover-glasses and cultures taken. At first it was thought that it could not possibly be a case of true diphtheria, as no source from which infection could have been brought could be thought of. After a few days the clinical symptoms and the appearance of the cultures left no doubt on the matter. As early as possible diphtheria antitoxin was obtained from Naini Tal and used once. This antitoxin was not injected a second time, as it was seen to be very old, and also at the seat of injection the skin, for about an inch and a half in diameter, showed dark red and black discoloration.

This serum had been issued in December 1901, and was consequently at the time two and a half years old. It was afterwards kindly retested by the manufacturers, Messrs Burroughs & Wellcome, and found to have lost 40 per cent of its potency. It had been kept in the comparatively cool climate of Naini Tal, so that it is probable that serum kept in the very hot climate of the plains of India deteriorates to a much greater degree and in a much shorter time.

On the 17th some fresh antitoxin was received from Bombay and a large dose injected, but it was evidently used at too late a stage of the disease, for the child sank and died on the 23rd.

The other children received an injection of antitoxin to protect them.

The discharge from the boy's nose continued for about three weeks longer, and he was given three separate injections of serum at intervals of three weeks.

The culture (Culture No. I.) obtained from the fatal case was tested on rabbits, bulls, fowls, cat, dog, and rats, and proved very virulent for these animals.

Cultures were taken on three occasions from the nostril of the boy— on the first occasion on 14th June (Culture No. 2); second, on 25th July; and third, on 31st August. The cultures taken on first and second times were found equally virulent as Culture I., but the culture taken on the third occasion did not kill a rabbit when inoculated subcutaneously.

When it was discovered that the case was undoubtedly one of true diphtheria every attempt was made to trace the source of infection, but we were unable to obtain any evidence that the contagium had been brought from any other infected area.

Lesions on Udders of Cows.—About two months previous to the outbreak one of the cows kept for supplying milk to these children, and which had recently been purchased and had a calf of a few weeks old, showed symptoms of illness. Her milk decreased, and she had a dry

husky cough which seemed to come from the throat. At times it looked as if there was some obstruction in the larynx or trachea. Frequently after coughing the cow kept her mouth slightly open for some minutes and held her head down and neck stretched, as if trying to dislodge something from the throat. I examined the mouth and throat several times, but was unable to diagnose the cause of the cough. There was no disturbance of pulse or temperature. The udder was not at this time examined. After three weeks the cow appeared to be quite well again, but still continued to cough slightly at times. Six weeks after the purchase of the first cow another newly calved cow was bought and put in the same stall. About two weeks after her arrival she developed symptoms similar to those which the first cow displayed.

Shortly after this the boy was affected with rhinitis. For some weeks before this occurred the children had been drinking fresh raw milk. Always previous to this the milk for their use had been carefully boiled, but at this time they were out of sorts, and it was thought that the fresh unboiled milk might be more palatable and nourishing.

On the 14th of June both cows were carefully examined. The udder and teats of the first cow showed marks of healed ulcers, dried vesicles, and crusts. Some of the crusts were the size of a pea, but most of them were much smaller. On the udder and teats of the second cow there were newly-formed vesicles, small ulcers, and a few small pustules.

Cultures on agar and blood serum were taken from the lesions on the udders of both cows. From the vesicles on Cow No. 2 a pure growth on agar was isolated. This showed a number of bi-polar stained bacilli with rounded ends, and other forms bearing a resemblance to the human diphtheria bacillus (Fig. 18). This culture and subcultures on blood-serum were tested on rabbits by subcutaneous inoculation, but, beyond causing a slight œdema at the seat of inoculation and a rise of temperature for two days, they had no effect. It was also non-virulent for fowls.

During the last week of October Cow No. 1 again went off her milk and appeared ill for some days. On examining her udder it was found to be covered with marks of healing ulcers, crusts, and a few vesicles. There were two or three scabs about the size of a pea, but the more numerous were not much bigger than a pin head. Cultures from the lesions gave no result. The cow was at this time disposed of.

Disease among the Fowls.—Early in May an epidemic had appeared among the fowls in Muktesar, which continued until the end of the rainy season in October. The disease appeared just before the rains, and was at its worst during the monsoon season, when there was a heavy rainfall almost daily. It was chiefly among the young fowls, but also some older hens and several ducks died. Some were fowls which had been some months in the place, but the greater number were birds which had been recently imported from Naini Tal and Almora. In all about fifty fowls died of the disease. The newly imported birds died in from ten days to three weeks after their arrival. The symptoms of the disease and the result of investigation are described later on.

A fact noticed in connection with the outbreak among the fowls and the illness of the cows was that in each instance the sickness followed after the turning up of subsoil from a depth of two to six feet. For some weeks before Cow No. 1 showed any symptom of illness, cuttings on a hillside close by were being made, and the ground near the cowhouse was covered with soil turned up from the depth of some feet. At the residence where the disease among the fowls first appeared the ground was being levelled for a site for an outhouse. Shortly after this work had been commenced deaths occurred among the fowls which had been daily picking over this ground. Some months after this, at another dwelling, excavations were being made to lay the foundation for an additional room to the building. A few weeks after this several deaths occurred among the fowls and ducks kept at this place, and which up to that time had remained healthy. Some three years previous to this a similar fact was observed when many fowls and ducks died at Muktesar from the same disease. At the residence where the cows became ill no loss among the fowls occurred. At this place the fowls were confined to an enclosed run, and did not get an opportunity of picking among the fresh soil. In October, when the rains ceased, the epidemic among the fowls disappeared.

Many cultures were taken from soil freshly turned up from various depths. On two occasions a pure culture of a bi-polar stained bacillus with rounded ends was obtained. This bacillus was morphologically identical with the organism constantly observed in the experiments with human and avain diphtheria. The cultures were tested on rabbits by subcutaneous inoculation, but were non-virulent. They caused slight swelling at the seat of inoculation and a rise of temperature for two or three days.

INOCULATION EXPERIMENTS WITH HUMAN DIPHTHERIA.

These experiments were carried out by the permission of the Imperial Bacteriologist, and with the assistance of Mr Max Kreebel, Head Laboratory Assistant. The object was to ascertain if any connection could be traced between the outbreak of human diphtheria, the sickness of Cows Nos. 1 and 2, and the epidemic amongst the fowls.

Culture No. I. was obtained from the false membrane in the throat of the fatal case of diphtheria. A swabbing was taken from the throat on the first day on which the illness was observed, and from this a pure culture of the Klebs-Löffler bacillus was obtained.

Culture No. II. was taken from the nose of a child who suffered from rhinitis. This culture was taken two days after Culture No. I. from a platinum loop soiled by discharge from the nostril, and from this a pure culture was isolated. The child had at this time been affected with discharge from the nose for about three weeks. These cultures were tested on rabbits and bulls, and proved fatal to both animals. Other cultures were taken from the boy's nose on 25th July, and again on 31st August, and a pure growth of diphtheria isolated. The second culture was virulent for rabbits, but the third culture seemed to have lost its virulency, and was not fatal to a rabbit when inoculated subcutaneously.

All cultures used for inoculation purposes were grown on blood-serum for forty-eight hours at a temperature of 37° C., and when injected subcutaneously were mixed with 2 to 3 cc. bouillon.

In obtaining material for cover-glass specimens and for cultures at the autopsies every precaution was taken to exclude extraneous organisms. The blood was in every instance taken from the heart by means of a sterile pipette.

Guinea-pigs were not used, as none could be obtained at the time.

Experiments on Fowls.

No. 1. A cock about three years old was inoculated, by scraping the throat, with Culture No. II. Showed no symptom of illness.

No. 2. A fowl about six months old was inoculated on same date and in same manner as Fowl No. 1. Died after seven days. For the first four days showed no symptoms, but on the fifth day was dull and not feeding; on the next day wings were drooped, and the bird seemed to be in a comatose state. There was difficulty in respiration.

Post-mortem.—Larynx, pharynx, trachea, and nasal cavities were covered with a fibrinous adherent membrane. There were a few small areas of necrosis where the mucous membrane of pharynx had been scraped. No other lesions were discovered.

Cover-glass specimens and cultures were taken from membrane and from heart's blood. In membrane numerous organisms were seen, some of which resembled typical diphtheria bacilli. There were a large number of ovoid bacilli and of bacilli with rounded ends, unstained centre, and deeply stained granular bodies at either pole.

In the blood the bi-polar forms alone were found. They were faintly stained, and of varying length and thickness. In some the central part was slightly stained, with polar bodies of a deeper colour. In some the granular bodies at the poles alone had taken the colour, and the other parts were unstained (Fig. 7, a). This bi-polar stained organism was constantly observed in the heart's blood and other tissues of animals which died after inoculation with human diphtheria virus (Fig. 7, a, b, c, d). In some instances these forms alone appeared (Fig. 6). In other cases they were associated with forms, more or less numerous, resembling the more typical shapes of the human diphtheria bacillus. In cultures this organism displayed a much more varied appearance. Few of the bi-polar shapes remained; some bacilli showed three or more deeply stained granular bodies, and many of the forms were identical with those of the Klebs-Löffler bacillus (Figs. 8 and 9).

A morphologically similar organism was constantly observed in the pseudo-membrane and tissues of fowls dead from avian diphtheria, and also was recovered from the tissue of animals dead after inoculation with avian diphtheria virus (Figs. 11, 12, and 15). Also in cultures this organism showed the same variety of forms more or less resembling the typical forms of human diphtheria bacillus (Figs. 13, 16, and 17).

From the blood of Fowl No. 2 a pure culture on agar was isolated. In this several different forms were observable: a short oval bacillus uniformly stained; a short oval bacillus with centre unstained; a bacillus with rounded ends and unstained centre; rods of varying

length and thickness uniformly stained, some with poles of deeper colour, and some showing three or more granular bodies; rods in club or flask shape, some with unstained central part.

Forms similar to these were obtained in cultures from the blood of rabbit, bull, and cat which died after inoculation with human diphtheria bacillus; also in cultures from blood of Fowl A dead of avian diphtheria, and from blood and tissue of animals dead after inoculation with material of fowl diphtheria. Compare forms in Figs. 9, 12, and 15.

Fowl No. 19 was inoculated subcutaneously with one loop (standard 2 mm.) of Culture No. I. Died in two and a half days. There was œdema at seat of inoculation, a white fibrinous deposit between the muscles, and subcutaneous tissue was much injected. Cover-glass specimens and cultures were taken from blood. The same forms were seen in blood as in case of Fowl No. 2. A pure culture was isolated from blood which was identical with culture from blood of Fowl No 2.

Fowl No. 20 received an intra-muscular inoculation of one loop from culture isolated from heart's blood of Fowl No. 19. Died in three days. *Post-mortem* appearances were similar to those observed in case of Fowl No. 19. Result of examination of blood and cultures from blood was the same as noted in Fowl No. 2 and Fowl No. 19.

Experiments on Bulls.

The bulls used were of hill-breed, weighing about 200 lbs., and under three years.

Bull No. 2885 was inoculated with a blood-serum culture (Culture No. II.), forty-eight hours old, in 5 cc. bouillon, subcutaneously behind the left shoulder. On following day temperature rose to 40°; there was extensive œdema at seat of inoculation and extending down the leg. The bull did not feed, and lay with his head round on flank. The next day the temperature fell rapidly, and he died during the night, about sixty hours after inoculation.

Post-mortem.—Much yellow gelatinous œdema at seat of injection. There were layers of white-coloured fibrinous deposit between the muscles, which on section showed a mottled appearance. The surrounding glands were enlarged, congested, and œdematous. At none of the autopsies were the lesions described by Klein observed, namely, large white fatty kidney, brown markings on liver, reddening of supra-renal bodies, and enlarged, engorged lymphatics of lungs. As a rule no internal lesions were recorded, except that in one or two instances the liver was fatty, and also the spleen enlarged and softer than normal. Cover-slips and cultures were taken from œdema, enlarged glands, and heart's blood.

Bull No. 2888 was inoculated with Culture No. I. on same date and in same manner as Bull No. 2885. On the following day the temperature rose to 41·3° C., and a large swelling appeared at seat of inoculation. On the next day the temperature was falling rapidly, and there was a white mucous discharge from nostrils. Died during night, about sixty hours after inoculation. The *post-mortem* appearances were similar to those described in case of Bull No. 2885. Cover-slips were taken from nasal discharge, œdema, and gland; also cultures from œdema and gland.

Bull No. 2299. Received subcutaneously one loop from Culture No. I. Same clinical symptoms were observed. Bull died five and a half days after inoculation. *Post-mortem* appearances were similar to previous cases. Cover-slips and cultures were taken from œdema, gland, and blood.

Bull No. 2300 was inoculated on same date and in same method as Bull No. 2299. Died six days after inoculation. Cover-glasses from œdema, glands, and heart's blood were kept.

Bull No. 2415. A month later, in order to test if Culture No. I. retained its virulency, this bull was inoculated as above, with one loop of Culture No. I. (sub-culture). Died six days after inoculation. Cover-slips and cultures were taken from œdema and heart's blood.

Bull No. 2406 on the same date received subcutaneously two loops of Culture No. I. Died in three days. Cover-slips and cultures were kept from œdema, heart's blood, liver, and spleen.

Bull No. 2440 three weeks later received two loops subcutaneously from Culture No. I. Died in two days. Cover-glasses and cultures taken from œdema and blood.

Bull No. 2390 on same date received two loops subcutaneously of Culture No. I. Died in sixty-four hours. No cover-slips or cultures taken.

Result of Microscopic Examination and of Cultures.

In almost every instance cover-slips were taken at the autopsies from the œdema, from an enlarged gland near seat of inoculation, and from the heart's blood, and at the same time cultures were made from the same source both on agar and on blood-serum.

In the œdema taken from the seat of inoculation, forms typical of the diphtheria bacillus were invariably observed, together with many of the less diagnostic bi-polar stained bacilli with rounded ends. In the œdema at some distance from the seat of injection the forms were less typical and less varied, and the bi-polar bacilli were in greater abundance. This fact was also noted in the case of animals which died after inoculation with fowl diphtheria. The forms recovered from the œdema at the point where the injection was made were of greater variety, and presented a more striking resemblance to human diphtheria bacillus than the organisms observed in œdema taken at a distance from this site or in the other tissues (Fig. 15).

In the gland the typical forms of human diphtheria were less numerous, and bi-polar bacilli were in greater numbers (Fig. 7, *d*).

In the blood bi-polar stained bacilli of varying length and thickness were constantly found. As a rule they were smaller but of similar shape to those seen in blood of rabbit dead after inoculation with human diphtheria (Fig. 6), and were also very similar in appearance to the forms seen in blood of Bull No. 2345 dead from fowl diphtheria. (Fig. 15, *b*). They were faintly stained, the body of the bacillus showing little or no colour, with polar bodies which had taken the stain somewhat better. There were seldom more than two or three in a field, and were difficult to distinguish without a high power and good light. The specimens stained with methylene-blue quickly lost their colour, and in cover-glasses kept two months the forms were no longer distinguishable. These bi-polar stained bacilli observed in the blood of bulls were morphologically identical with those obtained

ι·.ιι ·.κ· blood and tissues of the other animals, fowl, rabbit, cat, and
ω which succumbed to inoculation with human diphtheria bacillus
·· g. ·`, but were generally somewhat smaller. They were also
similar to the forms seen in blood and tissues of animals which died
after inoculation with fowl diphtheria virus. (Fig. 15. a, b).

From the œdema, gland, and heart's blood pure cultures were
isolated.

The cultures from œdema when grown on blood-serum generally
displayed the typical appearance of the Klebs-Löffler bacillus under the
microscope. The forms seen in cultures from the gland and from the
blood were less varied and less diagnostic. In addition to the simple
bi-polar bacilli observed in cover-slips, many other forms resembling
the human diphtheria bacillus appeared. It was invariably noticed
that in the blood of animals which died after inoculation with human
diphtheria, forms of the bi-polar stained bacillus alone could be
discovered microscopically, while in cultures from the same blood a
large variety of forms resembling more or less the Klebs-Löffler
bacillus appeared. Compare Fig. 6 and Fig. 7 with Fig. 8 and Fig.
9. Also that cultures on blood-serum were more typical than cultures
on agar. The same fact was noted in the experiment on animals
with fowl diphtheria. Compare Fig. 15 with Fig. 16 and Fig. 17.

Experiments on Rabbits.

Rabbit No. 1. Inoculated by scraping the conjunctiva of left eye
with Culture No. II. Died in four days. Cover-slips and cultures
were taken from the fibrinous exudate which covered the conjunctiva.
Numerous forms of the Klebs-Löffler bacillus were seen in this exudate,
and a pure culture was isolated.

Rabbit No. 2 was inoculated on same date, by scraping the throat
with Culture No. II. Died after thirty-six hours. There was slight
œdema round throat and some pseudo-membrane in trachea. On
agar a pure culture of the human diphtheria bacillus was obtained
from the false membrane.

Rabbit No. 93 was inoculated subcutaneously with one loop of
Culture No. I. Died in two days. There was an amount of gelatinous
yellow œdema at seat of inoculation, and injection of the subcutaneous
tissue. The liver was fatty, and there was about 20 cc. of fluid in
peritoneal cavity. Cover-slips and cultures were taken from the
œdema and from the heart's blood.

In the œdema numerous forms of the Klebs-Löffler bacillus were
seen, together with a large number of a small bi-polar stained bacilli,
and a number of small ovoid bacilli. In the blood a few faintly
stained bi-polar bacilli were seen in each field.

Pure cultures were isolated from the œdema and from the blood.
In the culture from the œdema the forms were diagnostic, but the
forms recognised in the culture from blood were much less typical.

Rabbit No. 92 was inoculated on same date and in same manner
as Rabbit No. 93. Died in forty-eight hours. There was much sub-
cutaneous œdema at seat of inoculation. The supra-renal bodies were
slightly redder than normal, and the liver was fatty. Cover-slips were
taken from œdema, liver, spleen, kidney, lungs, and heart's blood, and
cultures from the œdema and blood.

The small bi-polar stained bacillus was numerous in all the tissues

·examined. In the œdema there were many forms bearing a resemblance to the human diphtheria bacillus, but in the other tissues examined the bi-polar forms alone were seen.

The result of cultures from œdema and blood gave similar results to those obtained from Rabbit No. 93.

Rabbit No. 103 was inoculated subcutaneously on the abdomen with half a loop of Culture No. I. Died in thirty-six hours. There was much œdema at seat of inoculation, extending about three inches in diameter; also much injection of subcutaneous tissue. Peritoneum and part of the small bowels were deeply injected. Cover-slips were taken from œdema, liver, spleen, kidney, and blood, and cultures from œdema and blood.

In œdema a few small bi-polar bacilli were visible. The same forms were observed in blood, liver, spleen, and kidney. In liver and spleen the bacilli were somewhat larger, and in many fields they were lying in pairs. They varied much in length and also in manner of staining; some were deeply coloured and clearly visible, others showed only the polar bodies faintly stained (Fig. 7, c).

Pure cultures were isolated from œdema and blood, with results as described in Rabbit No. 93.

A cover-slip from a blood-serum culture twenty-four hours old, taken from blood, did not stain according to the differential stain of Neisser.

Rabbit No. 95 received a subcutaneous injection of ·5 cc. diphtheria antitoxin (2000 units in 3·5 cc.). On the following day it was inoculated, by scraping the throat, with one loop from Culture No. I. grown ·on blood-serum for forty-eight hours at 37° C. The rabbit showed no symptom of illness. Six weeks afterwards it was retested. It was inoculated as before, and died in thirty-six hours.

Rabbit No. 102 was inoculated on same date and in same manner as a control to Rabbit No. 95. Died after five days. In pharynx there were a few small areas of necrosis where the mucous membrane had been scraped. In larynx and trachea there was a small amount of pseudo-membrane. Cover-glasses and cultures were taken from blood. The results were similar to those before described.

Result of Microscopic Examination and of Cultures.

Almost every cover-glass specimen taken from œdema, blood, liver, spleen, lungs, and kidney contained more or less numerous organisms. The commonest form was that of a bacillus varying somewhat in length and breadth, with rounded ends, and having a deeply stained granular body, with centre faintly or not at all coloured. In the blood they were of smaller size, and larger in liver and spleen. In some instances they were lying parallel in pairs; some appeared to be joined end to end. Other forms were also observed—long bacilli with deeply stained poles and faint central staining; rod-like bacilli with tapering ends and a slight unstained space in centre; ovoid bacilli, some with clear central space; bacilli showing three or more granular bodies. In the œdema, when taken from the seat of inoculation, typical forms of the Klebs-Löffler bacillus were found and recovered in pure cultures; while in œdema taken at a point distant from the seat of injection mixed forms were seen, some typical, but the more numerous of the short and long bi-polar description. These were

identical in form with those observed in œdema of rabbits which died after inoculation with fowl diphtheria (Fig. 15, *c*).

The organisms obtained from the tissues of rabbits which died after inoculation with human diphtheria closely resembled in form those recovered from tissues of rabbits which died after inoculation with fowl diphtheria. (See Figs. 6 and 7, and compare Fig. 15, *a*, *b*).

In pure cultures three forms of organisms were constant—a small ovoid bacillus, some uniformly stained, others with a small clear central space; a bacillus with rounded ends, unstained centre, and deeply stained polar bodies; a short rod-like bacillus with rounded ends and uniform colour. Other forms were frequently present which bore a greater resemblance to typical forms of the human diphtheria bacillus—long bacilli with three or more deeply stained granules; rods with pointed ends and unstained space in centre; bacilli with a deeply stained granule at one or both poles, and some flask or club-shaped bacilli (Fig. 8, *a*). These forms are similar to those obtained from culture from blood of Rabbit No. 97, which died after inoculation with fowl diphtheria (Fig. 16, *a*, *b*).

Experiments on Cat, Dog, and Rats.

Cat No. 1 was inoculated by scraping the throat with one loop of Culture No. 1. During three weeks the cat showed no symptom of illness. After three weeks it received subcutaneously one loop of Culture No. 1. Died in four days. The subcutaneous tissue was much injected and infiltrated with a large amount of yellow gelatinous œdema. At seat of inoculation there was a layer of about a quarter inch of white fibrinous deposit. The liver was friable and fatty. In cover-glass specimens from œdema, blood, and spleen numerous bi-polar stained bacilli were visible, the body of the organism being faintly or not at all stained. The majority of these bacilli were very small, but there were other forms of varying length and thickness (Fig. 7, *b*). On blood-serum cultures from the œdema a great variety of forms with more resemblance to typical diphtheria bacilli were observed (Fig. 9, *b*). In agar cultures the forms were much less varied and less typical, and the bi-polar bacillus was more in evidence.

Rabbit No. 106 was inoculated subcutaneously with one loop of an agar culture forty-eight hours at 37° C. from œdema of Cat No. 1. It died after twenty-one hours. In cover-glasses taken from blood, spleen, and liver numerous bi-polar bacilli with rounded ends were seen in every field. They varied slightly in size, but almost all presented the same appearance of a central unstained space and deeply coloured granules at poles (Fig. 6). On blood-serum cultures from the heart's blood a large variety of forms appeared bearing a resemblance to the human diphtheria bacillus (Fig. 8, *a*).

Dog No. 2 was inoculated subcutaneously with one loop of Culture No. I. Died after three and a half days. The subcutaneous tissue was deeply injected, and round seat of inoculation there was a large amount of œdema. No cover slips or cultures taken.

Rat No. 1, about six months old, received subcutaneously half a loop of Culture No. 1. Died in three days. In cover-slips and in cultures from the œdema the same forms were observed as described in case of Cat No. 1.

Rat No. 2 (old) received subcutaneously half a loop of Culture No. 1. Died in four days. No cover-slips or cultures taken.

Rabbit No. 107 was inoculated subcutaneously with one loop of an agar culture forty-eight hours at 37° C. from heart's blood of Rat No. 1. Died in thirty hours. The result of examination of cover-slips from the œdema and heart's blood and of cultures from the same source were identical with those described in case of Rabbit No. 106.

EXPERIMENTS WITH FOWL DIPHTHERIA.

During the time in which the experiments with human diphtheria were being carried out some thirty fowls died of fowl diphtheria from spontaneous infection. In the following cases *post-mortem* examinations were made, cover-slips from the fatal membrane and tissues were examined, and cultures on various media were taken :—

Fowl A, a young hen about six months, was purchased among others for experimental purposes, and five days afterwards it died. The pharynx, larynx, trachea, and nasal cavities contained an amount of adherent pseudo-membrane. No other lesions were observable. In cover-glass specimens from the pseudo-membrane numerous forms resembling the human diphtheria bacillus were seen. The bi-polar stained bacillus with rounded ends and faintly coloured centre was the most common form in every field (Fig. 11). In the heart's blood similar forms were seen, but not of such variety, and the bi-polar bacillus was the most general (Fig. 12, *a*).

In culture on agar from the heart's blood a pure growth was isolated in which three forms were observed. An ovoid bacillus, some of which showed a small central unstained space, the bi-polar bacillus, and a rod-like uniformly stained bacillus. In cultures on blood-serum a greater variety of forms appeared. In pure cultures on blood-serum from the false membrane many forms were seen which in contour and arrangement resembled the human diphtheria bacillus (Fig. 13, *a*, *b*).

Fowl No. 3, a young hen, displayed signs of illness for a few days before death. Had been several weeks in the place.

Post-mortem.—On the hard palate and pharynx were several small areas of necrosis, and a small amount of fibrinous membrane in the larynx and trachea. Cover-slips from the membrane and from the necrotic mucous membrane showed forms resembling typical Klebs-Löffler bacillus, and also numerous bi-polar stained bacilli. In cultures on agar and blood serum from the membrane a pure growth was isolated in which the forms were morphologically identical with the human diphtheria bacillus. Cover-slips from a young blood-serum culture stained by the differential stain of Neisser.

Duck No. 1, about four months old, had been in the place for about three weeks. Had been ill for a few days, shewed symptoms of paralysis, was unable to move, and lost its voice. The pharynx, larynx, and trachea were covered with a pseudo-membrane. No other lesions were observable. Cover-slips from membrane and from heart's blood showed a variety of forms similar to those already described in case of Fowl A and Fowl No. 3 (Fig. 12, *b*, *c*). The bi-polar bacillus found in tissues of duck were larger than those found

B

in tissues of fowl. The cultures on agar and blood-serum showed forms of organisms similar to those in cultures from fowl.

Ducks 2, 3 and 4. These were cases similar to that of Duck No. 1, and the results of *post-mortem* and of examination of cover-slips and cultures were very much similar to those described in case of Duck No. 1.

Twelve other *post-mortem* examinations were made on fowls which succumbed to this disease. In every instance cover slips were taken from the false membrane and heart's blood, and in some cases from the lungs, liver, spleen, and kidneys. Cultures on agar and blood-serum were kept from the false membrane and from the heart's blood.

The majority of the fowls were young, but some were old laying hens. Most of the younger fowls had recently been brought to the place, and succumbed to the disease generally in from one to three weeks after being subjected to the contagion. They showed little symptom of illness until a day or two before death, when they appeared dull, with wings drooped, not feeding, and more or less in a comatose state. The fowls which had newly arrived were not at death in an emaciated condition, but some of those which had been here for several months, and in which the disease had run a more chronic course, were mere skeletons. In some cases small areas of necrosis on the mucous membrane of the mouth, and in most instances a pseudo-membrane adhering to the pharynx, larynx, and trachea, and often filling the nasal cavities, were found on *post-mortem* examination. Also in two instances when the oviduct was examined there appeared a similar pseudo-membrane lining the duct, and a similar organism was identified in this membrane. In some cases the lungs were congested, in others the liver was fatty, in some the spleen was somewhat enlarged, but there was no constant lesion which appeared to have any connection with the disease.

Description of the organism of Fowl Diphtheria.

Morphology.—In fowls which died of fowl diphtheria from spontaneous infection the constant form of organism found in the false membrane and tissues was a bacillus with rounded ends and deeply stained granular bodies at either pole, and a central unstained or slightly stained space. It is analogous in form to that of the hæmorrhagic septicæmias. It is non-motile and does not stain by the method of Gram. Pure cultures of this bacillus were isolated which proved virulent for fowls, bulls, and rabbits. The same organism was found in tissues of inoculated animals and recovered in pure culture. This bacillus frequently varied in length and thickness, and in cultures presented itself under a variety of forms bearing a strong resemblance to the different shapes observable in a pure culture of the human diphtheria bacillus. The youngest forms seemed to be that of ovoids, in many of which under a high power a central unstained space could be seen. In the blood of the rabbit and duck the bi-polar bacillus was larger than in the blood of the fowl, and in the blood of the bull they were somewhat smaller.

Cultures.—The organism grows well on all the culture media. It grows best and more luxuriantly on glycerine-agar. In young cultures on blood-serum the forms appear in greater variety and more resembling the human diphtheria bacillus. The method of growth is very

HUMAN AND AVIAN DIPHTHERIA.

COMPARISON OF GROWTHS ON GELATINE AT ROOM TEMPERATURE, 22° C.

	24 hours.	48 hours.	72 hours.	Examined Microscopically after 72 hours.	Stain by method of Gram.
(1) Culture No I.	Very little growth, small translucent colonies, margins indented.	Growth slightly increased.	Growth very slightly increased.	Typical forms of the Klebs-Löffler bacillus.	Stain by method of Gram.
(2) Culture No. II.	Similar to (1).	Similar to (1).	Similar to (1).	Typical forms of the Klebs-Löffler bacillus. Many involution forms.	Do.
(3) Culture No. II. Taken on third occasion. Non-virulent.	Similar to (1).	More growth than (1).	More growth than (1).	Typical forms of the Klebs-Löffler bacillus. Numerous small bi-polar and ovoid bacilli.	Do.
(4) Human diphtheria passed through Rabbit No. 92.	Similar to (1).	Very slight increase of growth.	No increase.	Small ovoid bacilli and numerous bi-polar stained bacilli.	Do not stain by method of Gram.
(5) Human diphtheria passed through Rabbit No. 93.	Similar to (1).	Similar to (4).	Similar to (4).	Similar to (4), also some forms resembling human diphtheria bacillus.	Do.
(6) Human diphtheria passed through Bull No. 2888	Similar to (1).	Growth much greater than (1).	Growth increased.	Small ovoids, rod-like bacilli, and bi-polar stained bacilli.	Do.
(7) Human diphtheria passed through Fowl No. 2.	Much more growth of colonies similar to (1).	Growth much increased.	Growth increased.	Bi-polar stained bacilli mixed with ovoid and rod-like bacilli.	Do.
(8) Avian diphtheria. Fowl No. A.	Similar to (7).	Similar to (7).	Similar to (7).	Similar to (7).	Do.
(9) Avian diphtheria. Fowl No. 8.	Very little growth, hardly observable.	Growth increased.	Growth slightly increased.	Bi-polar stained and ovoid bacilli, and some forms having resemblance to human diphtheria bacillus.	Do.
(10) Avian diphtheria. Duck No. 1.	Similar to (7).	Similar to (7).	Similar to (7).	Larger bi-polar stained bacilli mixed with ovoid-shaped organisms.	Do.
(11) Avian diphtheria passed through Rabbit No. 97.	Very little growth of small greyish colonies.	Much increased.	No increase.	Similar to (10).	Do.
(12) Avian diphtheria passed through Bull No. 2345.	Similar to (7).	Slight increase.	Slight increase.	Small ovoid forms, bi-polar stained bacilli, and some rod-like bacilli.	Do.

similar to that of the Klebs-Löffler bacillus, and the appearance of isolated colonies is identical. In blood-serum cultures twenty-four hours old a thin transparent coating was visible, at the border of which were isolated colonies of greyish colour and margin finely indented. On glycerine-agar, twenty-four hours, there was an abundant transparent growth. On plain agar, twenty-four hours, there was a similar growth, but somewhat thicker than on glycerine-agar. In glycerine-bouillon, twenty-four hours, there was a slightly opaque flocculent growth ; at the bottom was much sediment. After forty-eight hours the bouillon remained quite clear. In plain bouillon, twenty-four hours, rather more growth than in glycerine-bouillon ; little sediment at bottom and bouillon not dim. On potato boiled, and on potato boiled and alkalised, in twenty-four hours there was no observable growth. In forty-eight hours a few small white colonies appeared, which increased slightly in size, but after seventy-two hours remained stationary. Grows anærobically in glycerine-agar. Does not coagulate milk.

Inoculation Experiments on Fowls.

Several healthy fowls were inoculated with material from the false membrane, by scraping the throat, and also subcutaneously with blood from the heart, taken from fowls which had died from avian diphtheria. Also many fowls were inoculated with cultures from the blood and pseudo-membrane of diseased fowls, but except in one instance there was no result. Pigeons were treated in the same way but without result. In order to raise the virulency of the contagium, the method practised by Guerin was tried, namely, inoculating pigeons in the conjunctiva with some of the false membrane taken from an affected fowl. This also failed to have any effect.

Cock No. 1. This was an old bird, about three or four years. It was inoculated by scraping the throat and rubbing over the abraded surface an emulsion of pseudo-membrane taken from Fowl No. 8, after death. On the fifth day the throat was examined and found to be covered with numerous small spots of necrosis.

Material from the necrosed area was examined and found to contain numerous small bacilli with rounded ends and deeply stained polar bodies, and also many organisms of ovoid form. On the eighth day the cock showed signs of illness, did not feed, had difficulty in respiration, and the nostrils and mouth were filled with an adherent sticky membrane. Died on the eleventh day after inoculation.

Post-mortem.—The mouth and nasal cavities, also pharynx and trachea, were filled with a pseudo-membrane. The lungs were congested and œdematous, the liver was fatty. No other lesions were observed.

Cover-slips from the pseudo-membrane showed numerous organisms, among which bi-polar stained bacilli were most in evidence. Many forms were very similar to typical shapes of human diphtheria bacillus (Fig. 14, *a*). The same forms were observed in cultures on agar and blood serum. In cover-glass specimens from the blood the bi-polar stained, bacilli varying somewhat in size, were seen, a few in each field (Fig. 14, *b*).

No culture from the blood was taken. The forms observed in blood were morphologically identical with those seen in blood of fowls which died after inoculation with human diphtheria (Fig. 7, *a*).

Experiments in Rabbits.

Rabbit No. 97 was inoculated subcutaneously with 2 cc. of a bouillon emulsion of pseudo-membrane taken from throat of Fowl No. 8 after death. Rabbit died in twenty-four hours.

Post-mortem.—Slight œdema at seat of inoculation and injection of the adjoining tissues were the only lesions evident.

Cover-slips were taken from the œdema, spleen, liver, and heart's blood, and cultures from the œdema and blood. In the blood, liver, and spleen bi-polar stained organisms were seen fairly numerous in every field. They differed only in length and thickness (Fig. 15, *d*). These are in form identical with those observed in the blood and other tissues of animals dead after inoculation with virus of human diphtheria (Fig. 7).

A great variety of forms were obtained from the œdema. The bi-polar stained bacilli were most numerous, but there were many forms bearing a resemblance to typical shapes of the Klebs-Loffler bacillus (Fig. 15, *c*).

A pure culture on agar was isolated from the blood, and this when twenty-four hours old was almost entirely composed of the bi-polar bacilli. In older cultures and in sub-cultures a variety of forms appeared, resembling those found in the œdema. When transferred to blood-serum the forms became smaller and finer, and in shape and arrangement showed a stronger resemblance to the human diphtheria bacilli (Fig. 16, *a, b*).

Two fowls and one pigeon were inoculated subcutaneously with one loop of a culture from blood of Rabbit No. 97. This had no result.

Rabbit No. 104. Received subcutaneously one loop from an agar culture forty-eight hours old taken from blood of Rabbit No. 97. Died in fifteen hours. Subcutaneous tissue was much injected, and there was a slight amount of œdema at seat of inoculation. No other lesions.

Cover-slips from œdema, blood, liver, and spleen showed organisms identical in appearance with those seen in tissue of Rabbit No. 97.

Pure cultures on agar and blood-serum were obtained from the heart's blood which were in every way similar to those recovered from the blood of Rabbit No. 97.

Rabbit No. 101 was inoculated subcutaneously with 0·25 cc. œdema from Bull 2259, which had died after inoculation with avian diphtheria. Rabbit died in twelve days.

Post-mortem.—There was much œdema at seat of inoculation. About 200 cc. yellow fluid in peritoneal cavity. Liver fatty.

Cover-slips from peritoneal fluid, œdema, and blood showed the presence of numerous bi-polar stained bacilli, similar in appearance but somewhat larger than those from Rabbits Nos. 97 and 104. They were morphologically identical with the forms observed in case of Rabbit No. 106 dead after inoculation with human diphtheria (Fig. 6).

Pure cultures on agar and blood-serum were isolated from the peritoneal fluid and from the heart's blood. In these the same bi-polar organism was seen together with forms resembling the human diphtheria bacillus.

Rabbit No. 105 was injected subcutaneously with 0·5 cc. diphtheria

antitoxin (2000 units in 3·5 cc.), and on the following day it was inoculated subcutaneously with one loop of a blood-serum culture from blood of Rabbit No. 97. Died in fifteen hours.

Result of microscopic examination of tissues and of cultures similar to those described in case of Rabbits No. 97, No. 104, and No. 101.

Experiments on Bulls.

Bull No. 2345 was inoculated subcutaneously behind left shoulder with five loops of an agar culture forty-eight hours from blood of Fowl A. Bull died in four days. There was much œdema at seat of inoculation, extending down the fore leg. No other lesions observable.

Cover-slips were taken from the œdema, heart's blood, liver, and spleen, and cultures from the œdema and blood. In the œdema and tissues a small bi-polar stained bacillus was seen. They were much smaller and not so numerous as those found in tissues of rabbits inoculated with avian diphtheria. In some the granular body at one pole was much larger than one at opposite end (Fig. 15, b).

A pure culture on agar was obtained from the heart's blood, and in this a variety of forms were observed—numerous bi-polar bacilli and other forms resembling the human diphtheria bacillus. The forms were very similar to those recovered in culture from blood of Bulls Nos. 2406 and 2415 dead after inoculation with virus of human diphtheria (Fig. 9, a, c).

Bull No. 2259 was inoculated subcutaneously behind the left shoulder with 1 cc. œdema taken from Bull No. 2345. Bull died in four days.

Post-mortem.—At seat of inoculation there was extensive œdema under the skin and infiltrating the muscles. In pleural cavity there was about 500 cc. yellow fluid on either side. The peritoneal cavity contained about 1500 cc. yellow fluid. The apex of the heart was covered with a yellow gelatinous œdema.

Cover-slips were taken from the œdema, heart's blood, liver, and spleen, and cultures from the œdema and blood. In the œdema and tissues the same organism was seen as in case of Bull No. 2345. This bi-polar organism was slightly larger and more clearly stained, and also more numerous than in Bull No. 2345.

A pure culture from the heart's blood showed a variety of forms similar to those observed in culture from blood of Bull No. 2345 (Fig. 17, b).

The cat, dog, and rat proved resistant to inoculation subcutaneously with a bouillon emulsion of the pseudo-membrane taken from throat of affected fowls and with culture from blood of Fowl A and Fowl No. 8, and from blood of Rabbit No, 97.

DESCRIPTION OF PLATES I., II., AND III.

VIRULENT HUMAN DIPHTHERIA.

FIG. 1. Represents forms of the Klebs-Löffler bacillus in a cover-glass preparation obtained from the throat of fatal case. The throat was touched with a plug of sterile cotton-wool on the first day on which symptoms of diphtheria were observed, and from this cover-slips and cultures were taken.
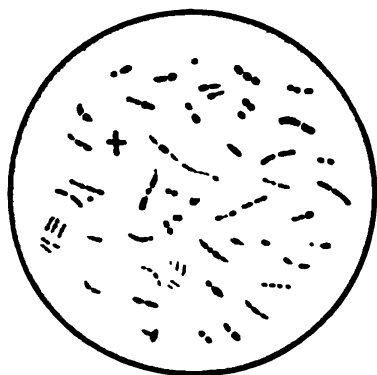
Fig. 1.
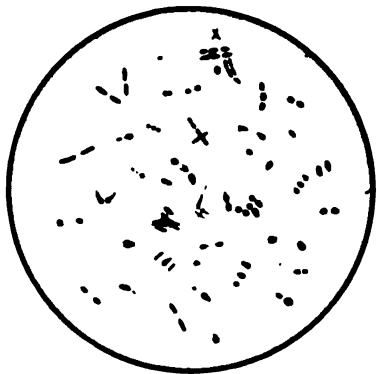
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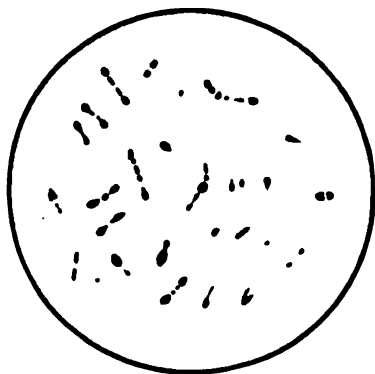
Fig. 3.
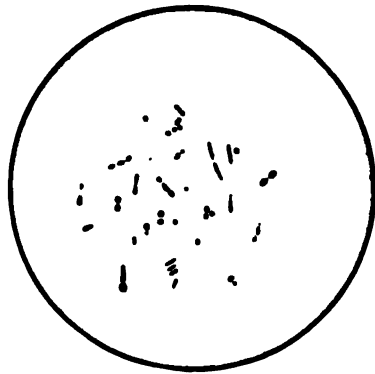
Fig. 4.
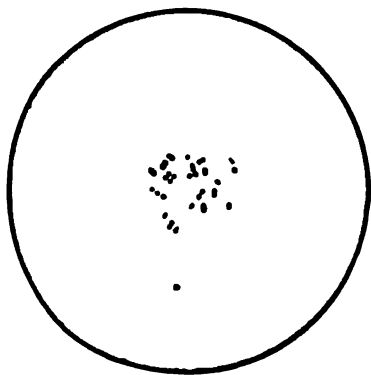
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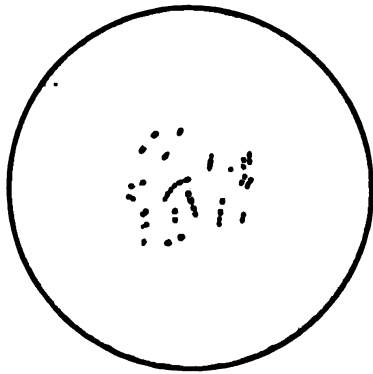
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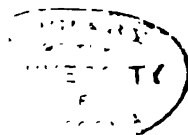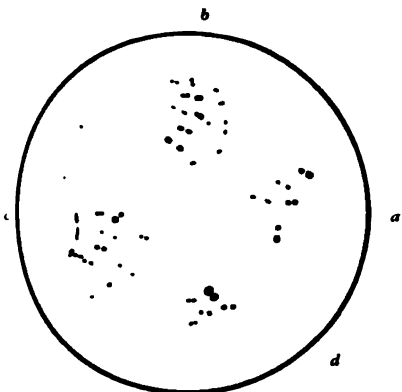
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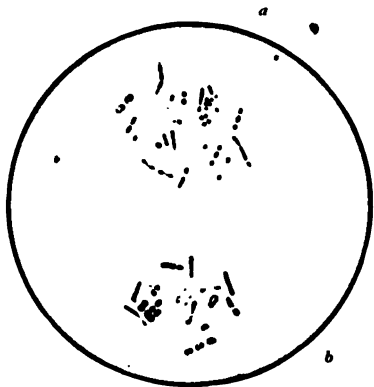
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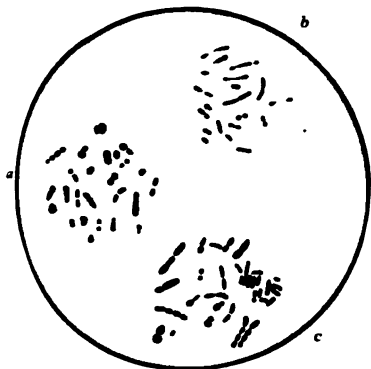
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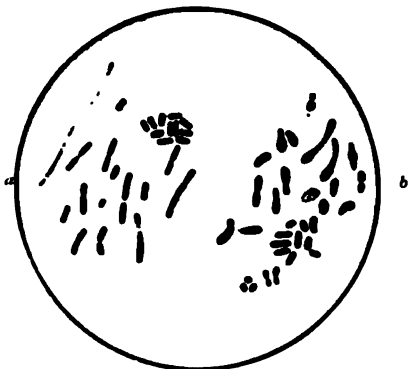
Fig. 10.
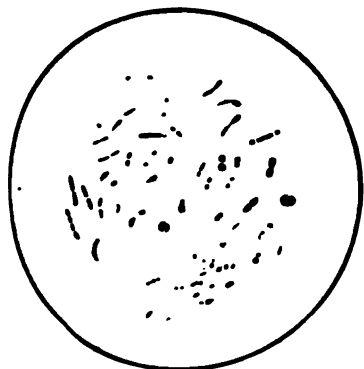
Fig. 11.
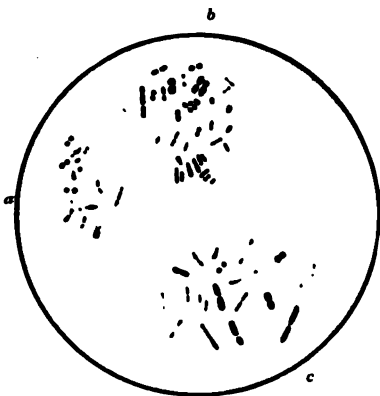
Fig. 12.
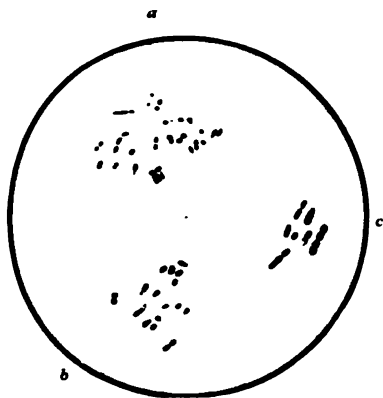
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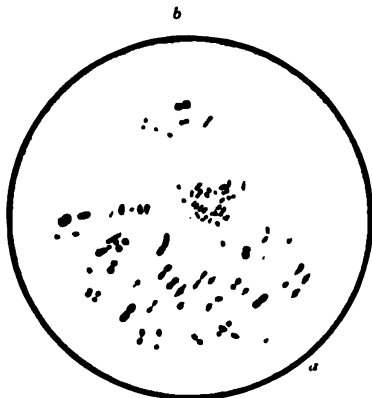
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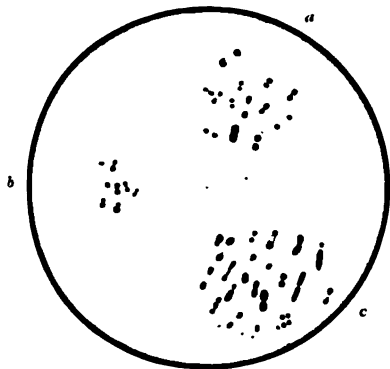
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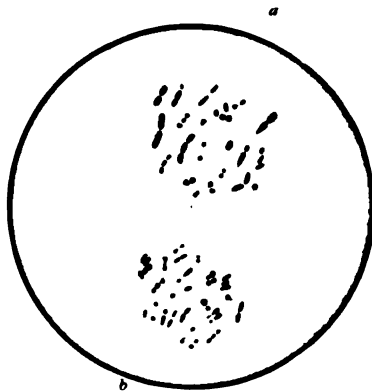
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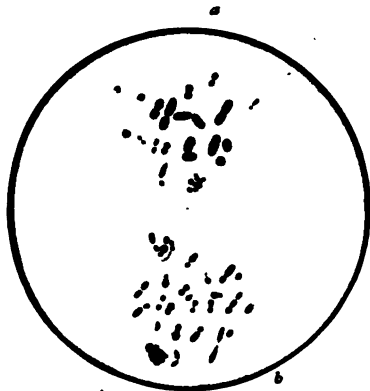
Fig. 17.
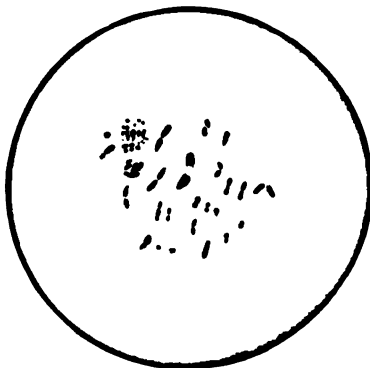
Fig. 18

FIG. 2. These are forms from a blood-serum culture twenty-four hours incubated at 37° C. The culture was taken from the throat of fatal case.

FIG. 3. Forms taken from the same culture as Fig. 2 seventy-two hours old.

FIG. 4. Represents forms seen in an agar culture incubated for forty-eight hours at 37° C.

FIG. 5. Forms from the same culture as Fig. 4, ten days old, incubated at 37° C.

FIG. 6. These forms were observed in blood of Rabbit No. 106. This rabbit died after inoculation with a pure culture from subcutaneous œdema of Cat No. 1, which died after inoculation with virus of human diphtheria.

FIG. 7. Magnified by 1500 diameters. (a) Forms in blood of Fowl No. 2. (b) Forms in subcutaneous œdema of Cat No. 1. (c) Cover-slip taken from the liver of Rabbit No. 103. (d) Cover-slip taken from an enlarged gland of Bull No. 2300. These animals died after inoculation with virus of human diphtheria.

FIG. 8. (a) Forms observed in an agar culture forty-eight hours from blood of Rabbit No. 106. (b) Forms in an agar culture forty-eight hours from the blood of Fowl No. 2.

FIG. 9. (a) Forms seen in a blood-serum culture forty-eight hours from blood of Bull No. 2406. (b) Forms seen in a blood-serum culture forty-eight hours from œdema of Cat No. 1. (c) Forms from a blood-serum culture forty-eight hours from blood of Bull No. 2415. These animals died after inoculation with virus of human diphtheria.

FIG. 10. (a) Forms observed in a blood-serum culture which had been taken from an old agar culture and left for twenty-four hours at room temperature, 22° C. (b) Forms from a blood-serum culture from blood of Bull No. 2300, kept for twenty-four hours at room temperature, 22° C.

AVIAN DIPHTHERIA.

FIG. 11. These forms were observed in the pseudo-membrane in throat of Fowl A, dead from avian diphtheria.

FIG. 12. (a) Forms in blood of Fowl A. (b) Forms from pseudo-membrane of Duck No. 1. (c) Forms from blood of Duck No. 1. Fowl A and Duck No. 1 died from avian diphtheria.

FIG. 13. (a) Forms seen in an agar culture twenty-four hours from the pseudo-membrane of Fowl No. 16. (b) Forms in an agar culture twenty-four hours from blood of Duck No. 2. (c) Forms in an agar culture twenty-four hours from blood of Fowl No. 4. Fowl No. 16, Duck No. 2 and Fowl No. 4 died from avian diphtheria.

FIG. 14. (a) Forms observed in pseudo-membrane of Cock No. 1. (b) Forms observed in blood of Cock No. 1. The cock died after inoculation with virus of avian diphtheria.

FIG. 15. (a) Forms from blood of Rabbit No. 97. (b) Forms from blood of Bull No. 2345. (c) Forms from œdema of Rabbit No. 97. Rabbit No. 97 and Bull No. 2345 died after inoculation with virus of fowl diphtheria.

FIG. 16. (a) Forms observed in an agar culture twenty-four hours from blood of Rabbit No. 97. (b) Forms observed in a blood-serum culture twenty-fours hours taken from the above agar culture from blood of Rabbit No. 97.

FIG. 17. (a) Forms taken from an agar culture from blood of Rabbit No. 97. This culture was kept for twenty-four hours at room temperature. (b) Forms taken from an agar culture seventy-two hours from blood of Bull No. 2359.

FIG. 18. Forms observed in an agar culture twenty-four hours taken from udder of Cow No. 2.

A CONTRIBUTION TO THE STUDY OF MASTITIS IN COWS.

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THE diseases common to the udders of cows have, for some unexplained reason, been rather neglected by the standard writers on veterinary science in this country, and, considering their importance in relation to the milk supply, this neglect is rather unaccountable. There is, however, a great deal of literature recording the researches of men of the veterinary profession on the continent of Europe. Their names include those of Nocard, Bang, Kitt, Jensen, Zschokke, Leblanc, Guillebeau, and others. Leblanc in 1901 issued a small book on the diseases of the udders of domesticated animals, which summarises the work of others and is an excellent exposition of the subject.

The purpose of the following investigation is to describe some of the inflammations of the udders of cows which are most commonly met with in veterinary practice. A series of cases of mastitis is described just as they emerged in the course of slaughter-house inspection, and without selection, except in so far that only cases in which an immediate bacteriological examination was possible were taken, *i.e.*, from two to eight hours after death.

The methods followed were : (1) to note the external appearances, and what may be disclosed by palpation ; (2) to describe the macroscopic appearances of the tissues on section ; (3) to record the result obtained from examining cover-glass films direct from the tissues ; (4) to describe the microscopic appearances of sections of the tissues ; and (5) to give the result obtained by cultures taken from the tissues.

After examining fourteen cases of mastitis it was found necessary to examine the normal udder for purposes of comparison. In each mastitis species of bacteria constantly recurred which had no pathogenic effect on laboratory animals. This suggested the possibility of their constant occurrence in the normal udder. Seven normal udders were examined, and for convenience these will be considered before the diseased ones.

I. NORMAL UDDERS.

CASE I.—Two quarters on one side were removed from the carcase of a cow for examination. The animal had been disused as a milker for about three months. The skin was thoroughly asepticised by means of lysol, alcohol, and æther, and a transverse section was made with a hot knife about 4 inches above the base of the teat and superior to the sinus. Four cultures were made and several cover-glass films. The films did not give very satisfactory evidence of the presence of bacteria.

Bacteriology.—The cultures, however, all showed the presence of bacteria. The first twenty-four hours' incubation at 37° developed only a few minute colonies on the two blood-serum tubes, but they grew well at room temperature. Two kinds of micro-organisms were isolated, a staphylococcus and a bacillus.

The *staphylococcus* colonies were greyish-white and slimy. This coccus has been repeatedly isolated, and in its growth on ordinary media it very closely resembles the staphylococcus pyogenes albus, but the cocci are larger, sometimes vacuolated, and sometimes they exhibit one deeply staining granule, and yet again both these features may be absent. This micro-organism is very commonly found in both normal and diseased udders.

The *bacillus*, when studied, appeared to be identical with the bacillus subtilis, except that it liquefied blood serum much more rapidly than did a culture of that bacillus obtained from a hay infusion.

Although the occurrence of bacteria in the normal udder was suspected, the actual finding of them came somewhat as a surprise. I was not then aware that they had been found by various Continental investigators. In order to verify the results of the second and all subsequent examinations of normal udders, cultures and films direct from the tissues were made by independent observers, for which my thanks are due to Dr W. T. Ritchie and Mr Watson.

CASE II.—In this case two quarters of one side were examined. (This procedure obtained throughout the series.) They were taken from the carcase of a cow which had been milking probably no less than eight months.

Experiment.—A guinea-pig was injected intraperitoneally with 2 cc. of the fluids exuding from an incision into one quarter. No result.

Films direct from the udder yielded no definite result.

Bacteriology.—Twenty cultures on various media were made from the tissues. Nine of these were made by Mr Watson, the assistant having charge of the bacteriological work of the laboratory, and the rest by myself. With the exception of five, all showed the presence of micro-organisms.

The colonies which largely predominated were those of staphylococci. A few of these were pale yellow, the rest greyish-white, profuse, and slimy. Next in frequency of occurrence came a fairly large sporulating bacillus. It is decolourised by Gram's method, but only feebly retains the stain. It is feebly motile. It has a marked enlargement in the centre indicating the growing spore. On agar streak culture, after twenty-four hours at 37°, it exhibits a profuse but delicate transparent growth. Magnified 50 diameters the colonies are raised, light brown, and non-granular. Gelatine is not liquefied. On blood-serum the medium is energetically liquefied, and when the process is complete there remains a grey stratum above the liquid. Milk is not coagulated, and its reaction remains neutral to litmus, alkaline to cochineal. Peptone is present. Old milk cultures are whey-like, with a thick white pellicle. This bacillus belongs to the genus tyrothrix, as described by Duclaux.[1]

CASE III.—The two quarters were taken from the carcase of a cow in full milk, which was slaughtered on account of leg fracture. Nine cultures were made from them—three by Dr Ritchie, four by Mr Watson, two by myself.

Bacteriology.—The three cultures made by Dr Ritchie were on the surface of agar. Two were made from a tissue section far from the sinus, and the third was taken from the fluids of a duct near the sinus. All three exhibited a very scanty growth of a pseudo-diphtheria

[1] *See* "Bacteriology of Milk." Swithinbank & Newman, 1903. Pp. 447-451.

bacillus in pure culture.   After forty-eight hours' incubation of bouillon sub-cultures at 37°, the reaction was still alkaline, and staining by Neisser's method exhibited no polar granules.

Of Mr Watson's cultures one agar and one bouillon were made from sections of the tissue near the sinus, and other two of like media were taken from a duct at the sinus.

The agar tube made from the gland tissue section was sterile. The bouillon from the same locality was slightly turbid after twenty-four hours at 37°, and films from it exhibited two different bacilli. One of these was short and motile, frequently formed chains and filaments, and was decolourised by Gram. The other was the pseudo-diphtheria bacillus. The agar culture taken from the duct exhibited profuse white colonies of staphylococci. The bouillon culture from the duct exhibited the staphylococci and the pseudo-diphtheria bacillus.

The two agar cultures made by myself from a tissue section in the upper third of one quarter were both sterile.

CASE IV.—In this case two quarters on one side were obtained from a neighbouring abattoir. There was no history of the cow to which they belonged, but they were apparently quite normal as regards size, palpation, and appearance on section.

Films made directly from section of the gland tissue and from ducts at the sinus exhibited only a very few isolated micro-organisms.

Bacteriology.—Six cultures from this udder were made by a fellow-worker and examined by me.

(1) *Bouillon.*—Loop scraping from the surface of the udder tissue on section. Sterile.

(2) *Bouillon.*—Loop scraping from a deep duct. Sterile.

(3) *Bouillon.*—Loop scraping from a deep section into udder. It exhibited a pure culture of the pseudo-diphtheria bacillus.

(4) *Blood - serum.*—Loop scraping from a section into udder. Exhibited one colony of a diplococcus.

(5) *Agar.*—Loop scraping from a section into udder. Exhibited one large white colony of staphylococci.

(6) *Bouillon.*—Loopful of milk from one of the main ducts. Exhibited four different micro-organisms. The most abundant form was the bacillus of pseudo-diphtheria, next a large bacillus resembling bacillus subtilis, then streptococci, and last a small short bacillus. This last mentioned bacillus when isolated and studied was found to be identical in its cultural features with the bacillus murisepticus of Flügge and Koch, with this important exception that it was not pathogenic to mice.

Experiments.—(1) A brown mouse was injected intraperitoneally with ·5 cc. of a bouillon culture, which had been incubated for twenty-four hours at 37°. No result.

(2) A tan mouse was similarly injected with ·5 cc. of a bouillon culture incubated for forty-eight hours at 37°. No result.

Four cultures were made by Mr Watson.

(1) *Bouillon*, from section into the gland tissue, remained sterile.

(2) *Agar slope*, from the same, exhibited one large white colony of staphylococci.

(3) *Bouillon*, from a duct near the sinus, exhibited streptococci in long chains and the bacillus of pseudo-diphtheria.

(4) *Blood-serum*, also from a duct, exhibited (1) a few of the small dry

grey colonies of pseudo-diphtheria, (2) one or two whitish colonies of staphylococci, each coccus exhibiting one deeply staining granule, and (3) one yellowish colony of a large bacillus. The latter bacillus in a day or two began to discolour and liquefy the medium.

CASE V.—As in the last case, two quarters of one side were obtained from a neighbouring abattoir. There was no history of the cow except that it was a dry cow brought there to be slaughtered and dressed as beef for the market. The quarters appeared to be normal in flaccidity and on section.

Nine films were made from one quarter, all from the milk sinus walls and the large ducts opening into the sinus, excepting one, which was made from a deep duct. Contrary to the usual experience, three of these films exhibited unmistakable chains of streptococci. That from the deep duct exhibited at least four chains, and the others one or two each.

Experiment.—A guinea-pig was intraperitoneally injected with 2 cc. of the fluids taken directly from a section into one quarter. No result.

Bacteriology.—Dr. Ritchie made two cultures on serum agar. One from a section of the gland tissue showed a few colonies of streptococci, the other from a duct exhibited streptococci, staphylococcus albus, and a bacillus resembling the pseudo-diphtheria bacillus, but which differed from those pseudo-diphtheria bacilli already found in that it exhibited polar granules when stained by Neisser's method.

Experiment. — A guinea-pig was subcutaneously injected over the lumbar region with 2 cc. of a bouillon culture of this pseudo-diphtheria bacillus incubated for twenty-four hours at 37°. No result.

Mr. Watson made four cultures. Two were made with loop scrapings from ducts and two from gland tissue, all from sections above but near to the sinus. Two were sterile and two exhibited streptococci.

My own cultures were five in number—four from loop scrapings of large ducts leading into the milk sinus, and one from a duct in that neighbourhood exposed by section. The four from the sinus ducts exhibited streptococci only, except that one also exhibited a whitish colony of the large staphylococcus. The fifth culture from the deep duct exhibited two colonies of a micro-organism not previously met with. This is a sporulating bacillus with an oval spore at one end like a drum stick. Its length is from three to eight times its width. It is feebly motile, the longer forms have undulating motion ; in some of the shorter it is rotatory. It stains by Gram's method, and is not acid-fast, but after staining in hot carbol-fuchsin it is not decolourised by thirty seconds treatment with absolute alcohol. Colonies in gelatine Esmarch tubes after forty-eight hours' incubation at room temperature are not visible to the naked eye, but magnified 50 diameters the surface colonies appear droplike, circular, and delicately granular. The deep ones are round and dark brown to black in colour. In three days liquefaction is apparent, when the deep colonies magnified 50 diameters present a seething mass of coarse granules in motion. In agar plates there is no appearance of growth to the naked eye in twenty-four hours at 37°, but after forty-eight hours at 37° small bluish colonies appear. Magnified 50 diameters the surface colonies are rounded, with one or two short blunt processes projecting

at the edge, and are delicately granular and homogenous ; the deep colonies are very dark and root-like, with short blunt processes projecting. Milk is not coagulated. In bouillon turbidity is not very marked. In fluid media the bacilli are frequently in pairs, often forming an obtuse angle. In old cultures of blood-serum there is partial liquefaction, the liquid having the appearance of water. It grows well at room temperature, and rather better anærobically than ærobically.

CASE VI.—In order to discover the period at which bacteria gained access to the udder, two quarters on one side were removed from the carcase of a heifer and examined. They were quite unexpanded. The animal was of the polled Angus breed and had only the two central permanent incisors erupted, and therefore was probably a little under two years old.

Mr Watson made four cultures from the gland tissue and the ducts exposed by the section.

I made six cultures from the sinus and ducts opening into it. All were sterile.

CASE VII.—Another heifer's udder was examined in the same way. The carcase from which the two quarters were taken was that of a shorthorn heifer with four permanent incisors erupted, or about two and a half years old. She carried a fœtus of about two months old. The udder was slightly expanded, and the sinus contained a clear thickish fluid

Eleven cultures were made, and all remained sterile.

General Considerations.

Most writers on this subject state that milk taken from the normal udder under aseptic conditions is sterile, and conclude therefrom that the interior of the gland is sterile. But this may only mean that the micro-organisms are associated with the walls of the ducts, and may not be withdrawn with the milk. As this is a matter of vital importance I will quote at some length from two recent writers on this subject. The following is translated from Leblanc's work already referred to.[1]

" M. Nocard has first established that the teat canal could serve as an avenue of entrance for pathogenic germs. This fact threw a vivid light on the pathology of mastitis. It established that the infecting agent has no need to penetrate the udder by abrasion of the skin, but that it has at its disposal a way of entrance always open and at hand, viz., the teat canal. This represents a capillary tube constantly moistened by milk, and consequently in excellent condition to serve as a means of conveyance for the micro-organisms which thus find 'a culture medium, and a road already made to reach the sinus' (Kitt).

" It thus becomes easy to understand the evolution of mastitis. The infected sinus represents the vestibule of the milk ducts in the same way as the pharynx represents the entrance to the respiratory and digestive systems. Just as bronchitis frequently succeeds pharyngitis, so mastitis often follows infection of the milk sinus.

[1] " Les Maladies des Mamelles chez les Animaux Domestiques," par P. Leblanc, 1901, p. 72.

Urethritis is a constant menace of orchitis and of cystitis, and the infection of the milk canal may have for the udder consequences as serious.

" It is no longer necessary to believe that the milk sinus of an udder which acts normally is exempt from germs. Most authors who have written upon bacteriology say that to obtain milk free from germs it is sufficient to draw it aseptically from the udder. That is absolutely an error.

" Of sixteen healthy cows examined under thoroughly aseptic conditions Guillebeau observed nine times infection of the teat canal. Zschokke has written that the sinus and the canal are constantly infected, like the mouth for example. This is certainly a manifest exaggeration, and the truth lies between the two. Six times out of ten we have found the milk sinus infected in the case both of the cow and the goat. Aseptic conditions were secured by means of superficial cauterisation, the udder being removed.

" In the majority of cases the habitual inhabitants of the sinus were found to be diverse varieties of staphylococci—S. albus, S. aureus, S. citreus. The variety which predominates is the aureus, but the three varieties can be found side by side.

" We do not attach any considerable importance to the presence of these germs in the vestibule of the milk ducts, because their pathogenic action has never been established. There is, however, room to think that if they are not the agents of mastitis they contribute to increase the gravity of the malady, and, perhaps, to cause the formation of mammary abscesses.

" Normally, then, the milk sinus is frequently inhabited by a considerable number of microbic colonies."

Otto Uhlmann,[1] a still later investigator, writes : " Until recently it was generally accepted that the secretion of the milk gland was free from bacteria, unless an ascending invasion through the teat canal caused a continuous infection of the secretion and gland itself with bacteria. That the milk is practically never germ free has been established by different investigators.

" The rationale of this has been given by bacteriologists who, like von Freudenreich, made use of fresh sterile milk in their researches, and who spared no pains to get if possible such milk. The natural sterility of milk, the work of Barthels, Ward, von Freudenreich, Burri, Boekhout and Ott de Fries, Backhaus and in more recent times Lux, is now definitely not to be regarded as a condition which is ever really present.

" With the establishment of this fact, however, comes the question by what avenue the bacteria gain access to the gland. It is self-evident that two possible avenues present themselves—the blood stream, and the teat canal, or both.

" It is the task of research to investigate the significance of these possibilities.

" I will now endeavour to state exactly what is the condition of the teat canal with regard to its bacterial flora, because the theory of invasion through the ductus papillaris has been up to the present time in favour. It has already been formulated as being acceptable in

[1] " Centralblatt für Bakteriologie, Parasitenkunde, und Infektionskrankheiten," 30th November 1903, p. 224.

view of the causal requirements of the case. Kitt [1] compares the
above canal to a capillary tube reaching from the exterior of the teat
up to the milk cistern. This column of milk forms a convenient high-
way for bacteria advancing from the contaminated outside surface to
the milk stored in the cistern."

Uhlmann found, by making serial sections of the teats of healthy
cows, goats, and sheep, that the teat canal is inhabited by many micro-
organisms, and records having found them in the amorphous plug
which sealed the external opening of the teat canal in a one-year-old
heifer.

It must be stated, however, that Kitt, in his work entitled " Bakterien-
kunde und Pathologische Mikroskopie" (1903), p. 389, says that
" The normal milk inside the udder, and at the moment of milking, is
absolutely free from micro-organisms." Further on, p. 395, he states
that by simply rubbing a potato culture of the bacillus phlegmasiæ
uberis upon the external opening of the teat, so that some of the
culture remained there, he produced the characteristic mastitis in a
cow. Here he repeats the statement that the teat canal is a capillary
column in which there is always milk, and consequently bacteria can
easily penetrate from the teat opening to the milk cistern. It would
thus seem that other bacteria, whether pathogenic or not, can
penetrate from the outside into the milk cistern by the same
channel.

There is no doubt that the blood stream brings bacteria to the
udder in some cases of tubercle and probably other diseases. But
that the teat canal is the avenue of advent |in a vast pre-
ponderance of cases is, I think, indicated by the observations herein
recorded.

It appears that, excepting those of central origin, the species of
bacteria found in a particular udder depend upon the accidental
presence of these species in its outside environment, and it is beyond
doubt that in those which I have examined a more exhaustive
examination would have revealed the presence of many more species
than are here recorded. Further, it is found that the localities inside
the udder which were richest in bacteria were the opening of the
main milk ducts into the sinus and the sinus itself. As the examina-
tion receded into the deeper ducts, and still more markedly when the
parenchyma in which no large ducts were visible, was examined, the
colonies on the culture media became progressively fewer.

There is reason to believe that these adventitious germs are almost
exclusively confined to the walls of the milk ducts, and that they exist
there as epiphytes rather than as true parasites. The fact that they
are so difficult to demonstrate in films taken directly from the ducts
and parenchyma shows that, relatively speaking, they are few and
isolated. This is also borne out by the fact that comparatively few
colonies appear on tubes of solid media.

Some of the germs found in the normal udder might well be derived
from the bedding-litter, the fodder, the fæces, and perhaps most im-
portantly of all, the hand and person of the milker. From these
sources also, in all probability, come the virulent micro-organisms
which directly produce mastitis.

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[1] " Lehrbuch der Pathologischen Anatomie der Haustiere," 2nd aufl., bd. I., 1900, p. 204-207.

II.—MASTITIS.

The following cases of mastitis are grouped into three classes, viz.:—

Tubercular, III. and V.

Purulent, VII. and VIII.

Parenchymatous, I., II., IV., VI., IX., X., XI., XII., XIII., and XIV.

The reasons for this classification will be stated after they have been described. The numbers given to them indicate the order in which they emerged.

The Tubercular Type.

CASE III.—This was taken from an Ayrshire cow about nine years old. The carcase exhibited generalised tuberculosis, and the disease had infiltrated the udder. The right half of it was markedly enlarged, particularly the posterior quarter. The skin covering these quarters was quite loose, and felt as if it intimately covered the indurated parenchyma, without the intervention of much subdermal areolar tissue. The indurated parenchyma felt stringy and ridged, but not nodular as might have been expected. This induration felt much more compact and hard than cases of mastitis of other origin. The most diagnostic tubercular feature was that the infiltration had accurately followed the original outlines of the quarter, so that the adjacent corners of the quarters on the lower surface could be distinctly felt where the transverse septum dividing the quarters, and the longitudinal septum dividing the two halves, approached each other. The ridge bounding the quarters along the longitudinal septum was also distinctly palpable. In ordinary mammitis the effusions and other inflammatory products practically obliterate these corners and ridges. Where the tuberculous induration becomes compact there is seldom any exudation between the skin and the indurated parenchyma. The non-nodular surface of the induration is not at all uncommon in tubercular mastitis. The compact induration did not extend quite to the base of the udder. The soft part had a firmer consistency than the normal tissue, but could not, if found apart from the compact induration, be diagnosed as tubercular. In a quarter or part of a quarter thus invaded, *i.e.*, before the interstitial tissue has become markedly developed and compact, the milk production is not arrested, but stimulated, and bacilli are passed out with the milk.

A section was made of the hind quarter from the teat to the base. The lower two-thirds of the cut surface disclosed the thoroughly invaded area in a solid block. The subdermal areolar tissue was not invaded. In colour this solid induration was only of a slightly deeper yellow than the normal gland tissue. Each lobule of acini was distinctly outlined, giving the surface a mosaic appearance, each piece in the mosaic being about the size of a split grain of pearl barley. The dividing lines between the lobules, however, were very faintly marked, and the pieces bulged very slightly upon the cut surface.

Films from this compact area were exceedingly rich in tubercle bacilli.

The area of softer consistency did not differ from normal gland

tissue in colour. Films made from this area showed not nearly so many bacilli as those from the compact tissue. This is the primary stage of the invasion, and by gradual transformation it passes into the secondary stage, which is the compact induration. Where the whole quarter is composed of the hard compact tissue the lesion has attained considerable age, and it is only then that calcification sets in. This third stage begins as a rule in the neighbourhood of the teat and advances upwards. It can be discovered by the grating sound emitted on section. When calcification is present the interlobular connective tissue is markedly increased, and can be seen by the naked eye, clear, white, and tough. The parenchyma then shows the unmistakable yellow tuberculous character. This advanced condition is rather rare.

Histology.—Sections of the soft tuberculous tissue show a considerable increase in the interacinous connective tissue, with active cell proliferation. There is a great increase in leucocytes present, but the outstanding feature of this section is the extraordinary increase in the number of the secreting cells lining the acini. This recalls and substantiates an observation by the late Professor Walley, that at the outset tuberculosis of the udder increases the secretion of milk. Few tubercle bacilli were found in this section.

A section of the compact tissue exhibits a vast increase in the amount of interlobular, and still more of the interacinous, connective tissue. The acini are often compressed to a mere slit. The abnormally numerous secreting cells of the acini are seen to be compressed laterally or obliquely, giving them a palisade appearance. In these solid areas are seen caseating foci, often with giant cells in the neighbourhood. The tubercle bacilli are abundantly distributed throughout these areas ; groups are seen inside the giant cells. The leucocytes and connective tissue cells are all markedly multinucleated.

Bacteriology.—Cultures were made from this mastitis, but the hopes of a pure culture of tubercle bacilli vanished when the micro-organisms incident to the normal udder appeared. These were the large staphylococcus with the cultural features of pyogenes albus, which was most common, and the bacilli of pseudo-diphtheria. The latter differed from those found in the normal udder, inasmuch as bouillon cultures gave a slightly acid reaction.

Experiment.—A guinea pig was injected intraperitoneally with 1 cc. of a forty-eight hours' culture of the pseudo-diphtheria. It died five months afterwards, apparently of a liver lesion, but I could not connect this with the above injection.

CASE V.—This was taken from a cow with generalised tuberculosis. The right half of the udder was enlarged and tubercular. The induration felt hard and compact, the skin was loose, and the surface of the induration felt ridged. Further description would simply be repetition of what has already been stated under Case III.

Cultures exhibited the white colonies of large staphylococci, and a bacillus resembling subtilis.

The Purulent or Streptococcal Type.

CASE VII.—This mastitis occurred in an old cow, the left half of the udder being affected. The two quarters were very greatly enlarged. The induration was in the lower part of these two

quarters, and felt tumefied, hard, and stringy, while the upper part was soft and flaccid. The lower parts exhibited irregular, smooth, bulging areas. A considerable amount of skin in the neighbourhood of the teats felt very hard and adherent. Posterior to the hind teat were one or two openings, which had been discharging pus. At the time of this examination the pus was inspissated. This was a chronic lesion.

A section made from the teats upwards, cutting through the hard adherent skin, showed that the hardness and immobility of the latter were due to the fact that it was about 2 inches thick from the epidermis to the parenchyma. This tissue was very dense, white, and homogeneous, and in one place a pus sinus about twice the thickness of a pencil had burrowed through it to the outside, where it discharged pus. Between this dermal wall and the parenchyma an amber serous fluid was found in the areolar tissue. Continuing the section upwards the milk cistern was found to be full of thick greyish-yellow pus, having a fetid odour. The walls of the milk cistern were of thick, white fibrous tissue, and were lined internally by a thin friable blue-black film of necrotic tissue.

A transverse section through the lower third of the two quarters exposed three or four pus sinuses and numerous discrete small abscesses of varying size. The parenchyma above this purulent region did not appear to differ from the normal, except that it was rather more flaccid and voluminous.

Bacteriology. — Films of pus disclosed a perfect chaos of micro-organisms among the degenerated cells and *débris*. The eye was at once arrested by chains of streptococci, which stained more deeply than the rest. Long badly-stained filaments were very noticeable. Occasional thick, deeply-stained bacilli appeared, and well-stained large micrococci occurring singly, or rather, hardly close enough to be described as forming groups. Closer examination disclosed myriads of badly-stained small cocci and bacilli.

Films from the tissue near the base of the udder exhibited only a large bacillus, sometimes in filaments, and a large coccus, often in pairs. No streptococci were found.

A film from the serum between the thickened skin and the paren-chyma exhibited chains of streptococci.

Histology.—Two small pieces of tissue were taken, one from the inferior suppurating region containing a minute abscess, and one from near the base of the udder. In sections of the first piece there was a considerable increase of fibrous tissue, which compressed the acini in every direction, and crowded the secreting cells close together. These secreting cells were abnormally large and œdematous. Short chains of streptococci were occasionally seen in the interacinous tissue, and groups of from four to six short bacilli. The latter were never observed inside the acini. The minute abscess exhibited chains of streptococci, up to about thirty members in a chain, and sometimes the terminal cocci were larger than the others.

Sections of the second piece, cut from the neighbourhood of the base of the udder, exhibited signs of commencing degeneration. The interacinous connective tissue seemed normal in amount, but the acini were rather larger than normal. The secreting cells were very œdematous, their nuclei were hazily stained, and their outlines in-

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distinct or lost. The only micro-organisms observed were occasional large cocci occurring singly and a few large bacilli.

Bacteriology.—The two predominating micro-organisms exhibited in the cultures were streptococci, the colonies of which were greatly in the majority, and short bacilli of the colon type.

The streptococci exhibited short chains, the members of which were minute in size. Their growth on serum was in minute colourless colonies, and they possessed the exceptional power of slightly eroding the surface of the medium, so that they appeared sunk beneath the surface.

CASE VIII. — This mastitis resembles the last described very markedly. The fore and hind quarters of one side were affected and were greatly enlarged. The lower third of each quarter was indurated, and bulged out in places, which proved to be the sites of large abscesses. Between this abscess region and the base of the udder the parenchyma felt flaccid. The skin of the areas round the teats was adherent, hard, and thickened, and proved on section to be dense and white.

On section the parenchyma of the lower third which was not destroyed by suppuration was greatly intersected by strands of white fibrous tissue, and the parenchyma often appeared as islands lying in a network of fibrous tissue. This was a chronic lesion of long standing.

Films made from the pus show myriads of micro-organisms, among which a small streptococcus is easily distinguishable as the prevailing form. Numerous staphylococci are also seen.

Bacteriology.—Cultures were made both from the tissue and from the pus. The prevailing forms were streptococci, but there also occurred large serum-eroding bacilli of the genus tyrothrix (Duclaux), a slender bacillus, and staphylococci.

Experiment.—Guinea-pig, 2 cc. of a bouillon culture of streptococci, incubated twenty-four hours at $37°$, were injected into the peritoneal cavity. Eight days after the injection the guinea-pig was killed. In the abdominal cavity was found a considerable quantity of serum mixed with blood, but there was no pus. Several blood clots were found in the neighbourhood of the lungs. The spleen was slightly enlarged, and the liver was enlarged and friable. The most marked lesion occurred in the areolar tissue between the skin and the abdominal wall. It was greatly increased in volume and of a dark red colour. The lesion extended over about one third of the inferior abdominal wall round the point of injection.

Cultures from this gross lesion were made, and the micro-organism was recovered.

The Parenchymatous or Hæmorrhagic Type.

CASE I.—*Lesion acute.* The cow in this case was killed a few hours after parturition. Both quarters of the left half of the udder were affected. They were greatly enlarged and pink in colour. The skin was loose, and a soft layer of tissue seemed to exist between it and the underlying induration ; the latter consequently felt smooth. On section there was a yellow gelatinous exudate in the areolar tissue between the skin and the parenchyma, and yellow serum flowed from it. The parenchyma was of firm consistency throughout, and serum and colostrum exuded from it, but no pus. On the cut surface large

continuous areas of the parenchyma were purplish in colour, while the remaining areas were still of the normal yellow colour. The purplish areas indicate hæmorrhage. The lesion was extremely acute.

Unfortunately, no films direct from the tissues were made.

Histology.—On microscopic examination the sections exhibit an increase in volume of the fibrous and cellular tissue, as compared with the normal. This is partly accounted for by the fact that the udder was in the colostral stage of lactation.

Colostral corpuscles are seen inside the acini in groups. Some of the acini are normal, but the striking pathological change is the frequent desquamation, partial or complete, of the secreting cells lining them. Sometimes the line of secreting cells is completely separated from its connective tissue base, but continues to maintain its character as a lining in a wavy irregular line, partly attached to the base, and partly free in the acinus. In other cases these cells are stripped away from the base, and are huddled in a mass inside the acinus.

Sections stained with fuchsin magnified 600 diameters exhibit very frequently groups of bacilli. These are seen for the most part inside the acini. In cases in which the desquamation is complete these microbes are seen mixed up with the *débris.* They seem able to penetrate through the base of the acinus into the interacinous connective tissue. Groups or masses of these micro-organisms are found only inside the acini. They appear to be the same size as the bacillus coli communis, their length being one-and-a-half times to twice their width, and some of them show bi-polar staining

The only other micro-organism observed is an occasional large coccus in the lymph spaces.

Bacteriology.—A bacillus of the colon group predominated practically to the exclusion of all others in all the cultures. It differs from the bacillus coli communis in that it does not coagulate milk, but on taurocholate glucose broth[1] with litmus it exhibits an acid reaction with formation of gas.

Experiment.—A guinea-pig was injected intraperitoneally with 1 cc. of a bouillon culture of this bacillus, incubated twenty-four hours at 37°. It died in about twenty-four hours from peritonitis, the only special features being a general faint redness and congestion of the mesenteric bloodvessels, and the presence of a little fluid in the abdominal cavity. The bacillus was recovered in a pure state, not only from the abdomen but also from the heart and spleen. Cultures exactly repeated the characteristics of the injected micro-organism.

CASE II.—This is an illustration of a more advanced case. It occurred in an aged cow. The fore and hind quarters on the left side were affected, and the induration extended throughout their volume. The skin was loose and the induration felt stringy and ridged. The quarters were much enlarged. On section a putrid odour was exhaled, and milk, blood, and serum flowed from it. The milk sinuses were sero-purulent cavities with dark grey walls near the base of the teats. The cut surface above the sinus exhibited a variegated appearance. The ground colour was grey with small abscesses of inspissated pus dotted over it. Several hæmorrhagic areas, usually oval in shape, were liver-coloured.

[1] Taurocholate glucose broth with Durham's fermentation tube. *See* Thompson Yates "Laboratory Reports," Vol. IV., Part I., 1901. Liverpool.

Films from the grey areas and the pus showed a profusion of microbes, the predominating forms being short bacilli. Next in numbers came minute streptococci in short chains, and a few large cocci.

The short bacillus occurred in a comparatively pure state in the liver-coloured, hæmorrhagic areas, and appeared to predominate in the other areas.

Histology.—The lesion is in the subacute and partially necrotic stage. The fibrous tissue is greatly increased in volume. Leucocytes have infiltrated most of the acini and sometimes red corpuscles. Everywhere there is evidence of blood extravasation. The acini, which are still intact, are few, and their secreting cells are enlarged and œdematous. Where the short bacilli have made their attack the acini and connective tissue are quite disorganised. The necrotic areas are frequent.

Bacteriology.—The predominating micro-organism in the cultures was a bacillus which, as far as observed, could not be differentiated from the bacillus coli communis.

Experiment.—A guinea-pig was injected intraperitoneally with 1 cc of a bouillon culture incubated forty-eight hours at 37°. The guinea-pig died in ten hours.

The *post-mortem* examination disclosed a diffuse peritonitis with a little abdominal fluid. The vessels of the small bowels were markedly injected. The spleen was very friable, the pericardium was injected, and the lungs congested. The bacillus was recovered from the thorax and abdomen wherever sought for. Cultures exhibited the same features as the injected micro-organism in every way.

CASE IV.—*Lesion subacute to chronic.* The left forequarter is affected. It is indurated throughout, but not much enlarged. The skin is loose, and the induration feels irregular in outline, being stringy and ridged.

On section the cut surface is greyish, exhibits many small discrete abscesses, about the size of peas, with inspissated pus. These are evenly distributed over the cut surface.

Films from the pus show micro-organisms in wonderful profusion and confusing variety. Next to minute cocci in numbers comes a short bacillus, then some larger thicker bacilli and large individual cocci, and finally a few short chains of streptococci.

Histology.—About three-quarters of the section area consists of acini smaller than normal, with connective-tissue bands surrounding small groups of acini and single acini. The fibrous tissue is increased in volume and has invaded the lobules, and by contraction it has diminished the area of individual acini. The occurrence of groups of leucocytes and of isolated groups of red corpuscles indicates frequent blood extravasations. The rest of the area consists of dull necrotic areas surrounded by broad irregular bands of connective tissue. In some places these bands show many deeply-staining cells, distinct from each other but in close proximity.

Magnified 600 diameters, these necrotic areas are seen to be collections of minute bacteria, staining indistinctly and mixed with *débris.* These areas are minute abscesses. Outside of the fibrous tissue bands bounding these abscesses, bacteria can be seen in the tissues in small groups, deeply stained and well-defined. Multi-

nucleated leucocytes are very numerous where these micro-organisms occur. The latter are mostly short bacilli, the length two and a half times the breadth, apparently of the colon type ; but there are a few chains of streptococci.

Bacteriology.—All of the bacteria above mentioned were recovered in the cultures. The only one which was isolated and cultivated was the tyrothrix filiformis, which was studied for the first time in this mastitis.

Experiment.—Guinea-pig. A serum culture of this tyrothrix filiformis was incubated for forty-eight hours, after which liquefaction was well begun. This growth was washed with bouillon, and 1 cc. of the mixture was injected intraperitoneally. There was no result.

This mastitis, like the preceding one, is one of the parenchymatous type which is passing from the subacute to the chronic stage. It is a mixed infection, but the colon bacillus is predominant.

The latter organism has completely invaded the quarter in the acute stage, producing induration throughout its parenchyma. In this vitiated tissue the pyogenic bacteria have found a suitable nidus, and their growth has resulted in the widespread abscess-formation present.

CASE VI.—*Lesion acute ; mixed infection.* In this case the two hind quarters were affected. They were enormously enlarged, and on one small area gangrenous. The skin was tense on account of the extent of swelling, but it was not adherent, except at the small gangrenous area and that immediately surrounding it. The gangrene was in the neighbourhood of the teat.

A section made between the affected quarters disclosed, in the subdermal areolar tissue of the inferior surface, a yellow gelatinous exudate in considerable quantity. There was also a considerable escape of sero-sanguineous fluid. A section of one of the quarters was accompanied by a further escape of the same fluid. The cut surface was of variegated colour, small greyish-red areas alternating with similar yellow areas, the former predominating in frequency. There was no admixture of pus in the escaping fluid, nor any sign of abscess-formation. It was an extremely acute lesion.

Films from the Tissue.—Those taken direct from the red areas exhibit a preponderance of streptococci and some short bacilli. There are also a few large bacilli. Films from the yellow areas show almost pure streptococci.

Histology.—Magnified 50 diameters. The interacinous and interlobular connective tissues are greatly increased in amount, often enormously so, and, judging from the looseness of their texture, they are engorged with serum and lymph. Lymph spaces in the connective tissue are proportionally enlarged. The lymph corpuscles are plentiful in amount and fairly normal in appearance. The blood vessels are all notably increased in lumen and engorged with blood, and in the red areas there frequently appear to be minute spaces about the size of acini filled with blood. The acini are mostly oblong or flatter than normal, as if they had given way to lateral pressure from the increased volume of the connective tissue. Their secreting cells are, in almost every instance, wholly desquamated, and are seen free in the acini in a degenerating condition.

Magnified 600 diameters, the micro-organisms which vastly pre-

dominate are the streptococci. They are seen in the acini in great numbers among the epithelial *débris* and milk traces. In the connective tissue the streptococci are not frequent, but occasional large bacilli are seen. Sometimes, however, in the connective tissue, where a lymph capillary is cut, there is an enormous amount of streptococci seen in the lumen and just outside of it in one direction. The chains are very short, being seldom over three members, but may extend to twenty. This indicates that these organisms use the lymph stream as their avenue of invasion. Sometimes large single micrococci are seen along with the streptococci in the acini.

The microscopic appearances of this streptococcus are more plainly seen in films taken from the tissues than in the tissue sections. The longest chains sometimes terminate in a coccus distinctly larger than the others. Sometimes the individual members of these long chains are double. In films taken from the red-grey areas, besides the streptococcus, bacilli are present, mostly in pairs. It appears as if the latter organisms are alone concerned in the production of hæmorrhage in these red areas, seeing they are absent in films from the yellow areas.

Bacteriology.—The cultures exhibit a variety of organisms, among which the streptococcus largely predominates. Short bacilli are the next most numerous, but they were not isolated in this instance. A large serum-liquefying bacillus made a vigorous growth in the original cultures. This micro-organism was isolated and studied, and is the same bacillus which was found later as an epiphyte in the first normal udder described, and is identical with the bacillus subtilis save in the nature of its growth on blood-serum.

Experiments.—(1) A guinea-pig was injected intraperitoneally with 2 cc. of a bouillon culture of the streptococcus, incubated for five days. No result.

(2) Another guinea-pig was injected intraperitoneally with 2 cc. of a milk culture of the streptococcus, incubated for two days. (Milk cultures were not coagulated till after four days' incubation.) The guinea-pig died in about forty-eight hours. The *post-mortem* examination exhibited pus free in the abdominal cavity, of a slimy semi-opaque nature, with yellow flakes throughout. The streptococcus was recovered from the pus in pure culture, and from the spleen.

CASE IX.—*Lesion illustrates healing and atrophy.* This udder was taken from a cow with generalised tuberculosis. Most of the organs were found to be infiltrated with the disease. The owner stated that about six weeks previous to the day of slaughter the cow "took a weed," as he termed the inflammation of the udder. Two quarters, the left fore and the right hind, were apparently normal. The right fore quarter was slightly indurated throughout, but not enlarged. The left hind quarter was shrunken to nearly one-fourth of its volume, that is, presuming its normal dimensions equalled those of the other three. It was much more indurated than the right fore quarter, and its induration was evenly distributed throughout.

This shrunken quarter was selected for examination because it differed so markedly from the cases already considered. During life, when this teat was milked, the cow showed evidence of pain and tenderness, and the fluid obtained was like serum slightly mixed with blood.

On section the colour was much yellower than that of the normal udder, and the lobules were more or less distinctly outlined. No other distinctive features were observed.

Films from this quarter exhibited no tubercle bacilli.

The cultures made remained sterile.

Histology.—Sections stained to demonstrate tubercle bacilli failed to exhibit the organism, or any of its effects upon the tissue, and other stains only exhibited a few groups of short bacilli, and large isolated cocci. The acini were smaller than normal, and often appeared compressed. The secreting cells in whole areas were entirely wanting, and where they occurred they showed great desquamation and disintegration. The connective tissue between the acini was increased in volume.

The shrinking up of the quarter must have been the result of the contraction of the interacinous and interlobular connective tissue, and the disappearance of a large proportion of the secreting cells of the acini. This contraction and atrophy of the inflamed quarter is, considering the ravages effected by the bacteria, the most desirable termination possible.

CASE X.—*Chronic enlargement without marked induration.* In this mastitis only one hind quarter was affected. The skin over it was everywhere loose, but the quarter was considerably enlarged. The induration was not so marked as usual, and manipulation seemed to reveal that the central mass was denser than the peripheral parts. This led to a suspicion of tubercular lesions.

On section it did not present any visible alteration from normal tissue, except that here and there occurred a thin streak of white fibrous tissue. No abscess or pus was visible.

Films direct from the udder were made with a view to discern tubercle bacilli, but none were found.

Histology. — Magnified 50 diameters. Sections show a marked increase in the interacinous connective tissue. The acini sometimes exhibit desquamation of their secreting cells. Magnified 600 diameters. The micro-organisms are not very plentiful in the section. Small oval forms of the colon type occur in groups inside the acini and among the secreting cells, and occasional short chains of streptococci are seen in the interacinous connective tissue.

Bacteriology. — Of six blood-serum cultures only one remained sterile. The most numerous colonies were those of streptococci, both the large and small forms, the latter greatly predominating. The next most frequent colonies were the slimy grey and white ones characteristic of the large micrococci of the normal udder.

Experiment.—Guinea-pig. 2 cc. of a bouillon culture of the large micrococcus were injected intraperitoneally. No result. So far as it goes, this experiment indicates that these epiphytic cocci are not enhanced in virulence when found in a mastitis.

Two kinds of large bacilli were found. One was of the genus tyrothrix (Duclaux), and the other resembled the bacillus subtilis.

The pathogenic bacteria in this mastitis exhibited a low degree of virulence, since so little alteration apparent to the unassisted eye had taken place during the time that must have elapsed while the white fibrous streaks were in process of formation.

CASE XI.—*Lesion acute, with partial necrosis.* The cow in this case

had died of septicæmia.   There was apparently acute inflammation of
the left hind quarter of the udder.   The teat of this quarter had been
partially removed by injury.   The skin of the teat and that covering
the neighbourhood of its base was thickened, but movable.   A yellow
necrotic area was visible on the teat, and the exposed and injured
tissue was probably the avenue of the infection from which the animal
perished.   The quarter was markedly and evenly tumefied.

On section there was no visible pus, but much fluid escaped.   The
tissue had a greyish appearance, with yellow necrotic patches here and
there about the base of the teat.

Films from the tissue.—Those made from the necrotic areas show a
marvellous profusion of organisms.   The most numerous are staphylo-
cocci ; next, large and small diplococci, streptococci in short chains,
and short bacilli with only occasional larger bacilli, and single large
cocci.   The short bacillus predominates in films from other parts of
the lesion, and is of two kinds, one resembling that of Cases I. and II.,
another always exhibiting only bi-polar staining.   .

Histology.—Sections exhibit great engorgement of the lymphatic
vessels and lymph spaces, and of the bloodvessels. •The interlobular
connective tissue is increased in volume, but this is owing to engorge-
ment by lymph, for the nuclei of the connective-tissue cells have
not increased with the volume of the tissue.   The acini appear rather
smaller than normal, as if they were being densely packed together.
Many are otherwise fairly normal in appearance, but most of them are
injured by the action of the microbes.   Desquamation is frequent, and
so is partial disarrangement of the secreting cells, but seldom are they
disorganised.   These cells are œdematous, and their enlarged con-
dition gives the picture a crowded appearance.

The tissue section is rich in organisms.   The most frequent are
short bacilli, which occur loosely arranged in groups, especially among
broken up acini.   There are some small masses of staphylococci, and
occasional short chains of streptococci.

Bacteriology.—The above micro-organisms were all recovered in the
cultures.   The short bacillus was found to be one of the colon group.

CASE XII.—*Lesion subacute to chronic, with advancing necrosis.*
No history of this case was obtained.   One quarter only was affected,
and it was greatly enlarged and evenly indurated throughout.   The
skin was loose and the induration felt ridgy.   On section the sub-
dermal areolar tissue appeared normal.   The cut surface was lobulated,
and the colour was greyish, this effect being produced by minute
areas of greenish-yellow inspissated pus upon a light brown ground.
The induration was not so intense as that of tubercle, and the affection
appeared to be subacute inclining to chronicity.

Films from the pus exhibited great profusion and variety of micro-
organisms, bacilli being almost as numerous as cocci.   The bacilli
were small ; some deeply, and some lightly, stained.   The lightly
stained bipolar bacilli were the most numerous, and occurred now for
the first time in this series (for description, *vide infra*).   The cocci
were small, single, and in groups.   Some short chains of streptococci
occurred, and a few large cocci.

Films from the tissue were not very much inferior to those from
the pus in number of organisms, but the latter were more clearly
defined.

On account of the lobulation seen on section of the quarter resembling tubercle, films from the udder and one from the tissue section were stained by the Ziehl-Neelsen method, but did not exhibit tubercle bacilli.

Histology.—The interacinous and interlobular connective tissue was markedly increased. The acini were compressed, and all of them showed partial or complete desquamation of secreting cells. Small abscess areas were very common. These tissues had a necrotic appearance, and micro-organisms swarmed in numbers which I have never seen equalled. Among them the unusual form above-mentioned was very common.

Bacteriology.—The growths on the original cultures were, as expected, of a very mixed character indeed. Films from them exhibited apparently all of the before-mentioned forms.

Microscopical appearance of the predominating bacillus.—It is a thick organism, its length being two or three times its width. It is usually pointed at both ends, in which case the staining is at both ends, but it is often rounded at one end, and stained at the pointed end. By fuchsin it is practically unstained except at one or both extremities. This feature is constant, and complete staining is only obtained by treatment with hot stain. In decolourising with dilute sulphuric acid the stain is completely given up, except a faint indication of colouring at the extremities. The best staining effect was obtained by first treating the film with absolute alcohol and then with cold fuchsin. By this method stained areas at the extremities amounted to about two-thirds of the organism, leaving one-third in the centre unstained. A large proportion of individuals are distinctly curved when in chains, which is very frequent in bouillon cultures. They are not exactly joined end to end, but the succeeding individual is attached to the convex surface near the point of its predecessor, the point itself being free. They are extremely motile, most of them darting with a fish-like motion, while a few show rotatory motion. Spores were not observed. In staining by Gram's method only the ends retain the stain, and that very faintly indeed. Curved forms are seen mostly in liquid media, not in the tissue.

Cultures.—Agar, twenty-four hours. Circular colonies raised above the surface, rounded on the top, but more flat than dome-like. Growth less vigorous than in bouillon.

Bouillon, twenty-four hours. Distinct turbidity, no collarette, and very slight deposit. The individual organism is larger than in agar, chains as already described being very common. Filaments are also common. They are distinctly wavy, resembling a spirillum, and the length from the summit to the depth of the wave is exactly that of the ordinary microbe. The turning point of the wave is more pointed than rounded. Occasionally a small, fully-formed bacillus is seen in the middle of the filament. The filaments mounted in bouillon exhibit immense activity.

Milk. No coagulation, and only slightly thickened in old cultures.

Taurocholate glucose broth, with litmus, is turned red in two days, but no gas is formed. Reaction acid.

Gelatine stab. The medium is liquefied. In two or three days there is a sack-like projection down the needle track from the surface. The liquid gelatine has a whitish-green appearance.

Experiment.—A guinea-pig had 2 cc. of a bouillon culture, twenty-four hours incubated, injected intraperitoneally. In eighteen hours it was found dead. Little change was observed on *post-mortem* examination, but a large amount of sero-sanguineous fluid was found in the peritoneal cavity. Films of this fluid exhibited the micro-organism injected, but rather sparsely distributed. They were plumper in form than any I had yet seen, and resembled a grain of barley in shape.

Cultures of the organism isolated from this fluid gave the same results as those already described.

This micro-organism exhibits features which relate it to the vibrios, notably the motile chains of curved forms, and the spirilliform filaments seen in bouillon.

CASE XIII.—*Lesion acute.* The cow from which this mastitis was taken had not yet shed its placenta, and was killed the day after parturition. The whole udder was enlarged and tense, which is often, in such a case, the normal condition. But one hind quarter was very specially so, and the skin of that quarter seemed thickened, but was easily movable.

On section of this quarter a great quantity of sero-sanguineous fluid flowed out. The cut surface exhibited a number of red areas on a normal coloured ground. The lesion was very acute. There were no purplish nor grey areas, nor was there any pus.

Films from the tissue serum.—There was not, relatively speaking, a great number of micro-organisms observed, and these consisted mainly of micrococci of three varieties. The vast majority of them were the large cocci common to the normal udder. They seemed to be in active proliferation, showing very frequent transverse segmentation. They occurred in groups and sometimes in masses, often they took the form of chains of three and two, and frequently the form of tetrads. Occasionally groups of smaller staphylococci were seen, but only one or two chains of streptococci. A few isolated short bacilli were noted, and one or two large bacilli. The large coccus was the predominating organism.

Histology.—The connective tissue between the lobules of acini was increased, but the tissue appeared loosely arranged, leaving frequent spaces in it. The interacinous connective tissue showed a wonderful wealth of leucocytes and connective-tissue cells, but this may be normal during the colostral period, though part of the excess was no doubt due to the irritation of the pathogenic microbes. The acini were to a large extent quite uninjured by microbes. The contents of the acini appeared to have greater consistency than normal milk, and in most cases contained round deeply-stained colostral corpuscles. There appeared to be great differences between the normal acini. In some the interacinous area was plentifully dotted over with the colostral corpuscles, and the secreting cells seemed at rest.

In others the secretion of colostrum was manifestly active. In these latter the secreting cells were about three times the normal length, projecting towards the interior of the acinus. They had an irregular palisade appearance. The large nucleus proper of each cell was generally situated near the base of the acinus, while at its distal extremity it was ragged and irregular from the detachment of colostral corpuscles. These on leaving the parent cells were enveloped by a narrow zone of protoplasm. The length of the secreting cells in the

active acini was such that the central area was diminished at least by half.

The micro-organisms seen in the tissues were not so numerous as might have been expected. Short bacilli, loosely grouped, were seen occasionally inside the acini, and were the cause of partial desquamation of the secreting cells, where they occurred. Cocci were not seen except of the large variety in isolated instances. The same may be said of large bacilli which were mostly club-shaped.

Bacteriology.—In all the cultures short bacilli occurred and quite predominated in number of colonies. Next in number of colonies came the large cocci of the normal udder, and, lastly, small discrete colonies of streptococci in short chains of very small numbers. The predominating short bacillus was not studied exhaustively, but it offered no special features which are not common to the colon group so far as observed.

CASE XIV.—*Lesion acute.* In this mastitis the left fore and hind quarters were attached. They were greatly enlarged, the induration was evenly distributed throughout, and the skin, though not thickened nor attached, was tense owing to the swelling. The hind quarter was selected for examination.

On section not much fluid escaped. In the subdermal areolar tissue a considerable amount of yellow gelatinous exudate was found. This was situated over the lower third of the quarter, especially in the neighbourhood of the teat. The cut surface exhibited areas of yellow and red, which were very sharply defined. The red or hæmorrhagic areas were most frequent towards the teat, but extended throughout the quarter. Sometimes these hæmorrhagic areas were large and continuous, and sometimes they were like red blotches on a yellow ground. Both red and yellow areas were indurated to the same degree. The milk reservoir was filled with a pus-like fluid. No pus was visible in the solid tissue. The lesion was a very acute one, and, after the cut quarter had lain for several hours, a great deal of serosanguineous fluid was found to have exuded.

Films direct from the tissues: *hæmorrhagic areas.*—The vast majority of micro-organisms are small short bacilli. One or two small masses of small cocci occur.

Yellow area.—Still the short bacilli are in the majority, but not in such great numbers as in the red area. Streptococci are common, in chains of from two to about twelve members. The large cocci occur occasionally. The main difference between the red and yellow areas is that the red contain the short bacilli in enormously greater numbers, and in seemingly greater vigour, judging from the depth of the staining.

Histology: *tissue sections from red area.* The impression given is that the lesion is very acute. Connective tissue cells have greatly increased in number, and in parts lymph spaces are very common indicating engorgement. The cut bloodvessels are enlarged in lumen, and in one or two areas of the section there is extravasation of blood. The acini, as a rule, show partial and often complete desquamation of secreting cells. Many areas are simply confused masses of connective tissue and secreting cells.

Magnified 600 diameters. The short bacilli are very numerous in the broken up acini. Their method of attack is seen in those are you

entirely broken up. They occur in streak-like multitudes on one side of the acinus, between the layer of secreting cells and the connective-tissue base. This explains why so large a proportion of acini are completely desquamated.

Bacteriology.—In all the cultures the predominating microbe was the short bacillus, which was found to belong to the colon group. The other micro-organisms recovered were streptococci, large staphylococci, and a sporing bacillus. The only distinctive feature which the colon bacillus offers is the appearance of involution forms in subcultures. The bacilli become vacuolated at one or both ends.

Experiments.—(1) Guinea-pig, 2 cc. of a bouillon culture of the colon bacillus, after twenty-four hours' incubation at 37°, was injected intraperitoneally. No result.

(2) Rabbit. It had the same dose of the same culture intraperitoneally. No result.

(3) Rat (white). It was injected intraperitoneally with ·5 cc. of the same culture. It died in about thirty-six hours. *Post-mortem* examination exhibited a small amount of fluid in the abdominal cavity. The border of the liver was livid. No morbid changes were observed in the thorax.

The bacillus was recovered from the abdominal fluid, the liver, and various parts of the thorax.

GENERAL CONSIDERATIONS.

In attempting to generalise upon the examination of fourteen cases of mastitis, it is fully admitted that types of the disease may occur which have not been observed in this series.

So far as our examinations go, they indicate that all udders of cows, both normal and diseased, have a bacterial flora. In the normal udder these bacteria are non-pathogenic. They appear to exist as epiphytes in the milk ducts. But, when the vitality of the udder is greatly lowered, such of them as are capable of pathogenic action can induce mastitis.

The variety of micro-organisms found in normal and diseased udders indicates that the bacteria found there depend wholly on the accident of their advent from the external environment, and upon their power to maintain their existence either as epiphytic or pathogenic forms. The occurrence of the vibrio in Mastitis XII. leads to the belief that there is probably an immense variety of pathogenic bacteria which are capable of producing mastitis, and that new types of the disease will be constantly emerging.

Classification.—The cases of mastitis considered in this series have been described under three groups: tubercular, purulent or streptococcal, and the parenchymatous or hæmorrhagic groups.

Tubercular mastitis.—Mastitis due to the invasion of the tubercle bacillus is specific and distinctive enough to need no further notice than reference to the cases described, numbers III. and V.

The purulent type.—This is exemplified in cases VII. and VIII. These are characterised by great thickening of the skin and subdermal areolar tissue, and by large pus sinuses, frequently connected with each other, and having very thick connective tissue walls with an internal lining of false necrotic membrane, blue-black or brown in colour.

This condition begins in the lower third of the quarter, and has a slow upward invasion. The upper stratum of parenchyma, which is uninvaded by purulent processes, is very soft and increased in volume through passive œdema. The predominating micro-organisms, which appear to be the cause of these changes, are streptococci of a low degree of virulence.

The parenchymatous or hæmorrhagic type.—Cases of this type are acute and subacute, the latter sometimes terminating in necrosis and discrete abscesses. This type is exemplified in cases I., II., IV., VI., IX., X., XI., XII., XIII., and XIV. The distinctive features of this type are the rapid and complete invasion of the whole quarter or quarters effected, diffuse or local hæmorrhages throughout the affected area, enlargement and induration due to infiltration of the interacinous and interlobular connective tissue by lymph, and in subacute and chronic stages this tissue is increased. In all the cases I have described under this type diffuse and local hæmorrhages were a constant feature. The micro-organisms which predominate in these hæmorrhagic areas are bacilli of the colon type. They differ from each other in some of their cultural features, and in their pathogenic effect upon laboratory animals. Esherich, in his investigations into the bacteria found in the intestines and fæces of healthy children, found a group of colon bacilli presenting similar variations in their morphological and biological features. Two American investigators, Booker and Jeffries,[1] in their study of summer diarrhœa in milk fed infants, obtained similar results with regard to bacilli of the colon type. They found several varieties of bacilli closely resembling Esherich's bacillus coli communis, some of which were rapidly fatal to laboratory animals, and some of which were not.

In Case VI. the predominating micro-organism is undoubtedly a streptococcus. The reason of its inclusion in the parenchymatous group is that the streptococcus, which was of unusual virulence, had, along with the colon bacillus, completely and rapidly invaded the two hind quarters, producing induration throughout and local hæmorrhages. The latter are undoubtedly due to the action of the bacillus of the colon group.

In sub-acute cases of this type small discrete abscesses with inspissated pus are invariably present, and sometimes one or two may reach the size of walnuts. Still the type is sharply distinguishable from that designated purulent by the induration existing throughout the entire quarter. Sometimes the necrotic changes are so widespread that the hæmorrhagic areas are obscured.

Mastitis in cows has an important bearing in relation to the milk supply, but it is difficult to obtain even an approximate estimate of the proportion of diseased udders which may effect it.

Swithinbank and Newman,[2] when writing on this subject, quote as the opinion of Professor M'Fadyean, in agreement with many other authorities, " that about 2 per cent. of the cows in the milking herds in this country are thus affected " (*i.e.*, have tuberculous udders). The question of tubercular contamination of milk from this source has already been so often and carefully studied that I propose to confine

[1] "Transactions of the International Medical Congress," Ninth Session, Vol. III. ; also "Archives of Pediatrics " Vol. VII.
[2] " Bacteriology of Milk," 1903, p. 215.

myself to the risks of milk infection from mastitis of other than tubercular origin.

There is no doubt that the percentage of cows suffering from mastitis of other than tubercular origin is very much higher than 2 per cent. The same authors give a table of the results of periodic veterinary inspections of the milch cows stabled in the metropolis which were carried out by the London County Council from 1899 to 1903. From this table it appears that, omitting actual and suspected cases of tubercular mastitis, the percentage of which was very low, the percentage of diseased udders varied from 13·2 to 4·8. The list of lesions enumerated are : " subjects of acute mastitis, affected with chronic induration of the udder, atrophy of one or more quarters, injuries, abscesses, simple eruptions, strictures and obliterations of milk ducts, and hypertrophied udders without induration." It is added that " these figures included cases of recently recovered udder disease, and a few cows removed by owners to avoid infection."

Partial restoration is the most that can be hoped for in a quarter or quarters which have been the seat of inflammation. The milking value of such a quarter is invariably reduced on account of the ravages of the causal micro-organism in the parenchyma. In such cases as have yielded to treatment to an extent which appears to justify the use of the milk for human food there may be grave risk to the consumer. Such udders are a probable source of the colon bacilli and streptococci so often found in milk, and incidentally of pus cells. These micro-organisms I mention because they are the commonest pathogenic bacteria found in parenchymatous and hæmorrhagic mastitis, but the infection is mostly a very mixed one. These bacteria and some of the tissue *débris* are largely eliminated through the milk, and it is this period of elimination which constitutes the danger, for of necessity it must be a very indefinite period.

With a view to connect the pathogenic effect of these bacteria on the consumers of milk, the following is quoted from Swithinbank and Newman, pp. 364, 365 : " That epidemic diarrhœa is caused by the bacillus coli either alone or in conjunction with other organisms, has been held by a number of authorities. Cumston, who investigated thirteen cases of the disease, concluded that bacillus coli associated with streptococcus pyogenes was the chief pathogenic agent concerned, and he claims that the virulence of bacillus coli is exalted by the association. Lesage also formed the opinion that the disease was due to bacillus coli, and investigated the agglutinative properties of the serum of children suffering from epidemic diarrhœa on bacillus coli isolated from the intestine. He obtained a positive result in forty out of fifty cases, and the serum of each of these forty cases also agglutinated samples of bacillus coli from thirty-nine other children seized with the same disease. Some of the most recent work on the relationship existing between bacillus coli and epidemic diarrhœa has been done by Delépine, who examined milk in the outbreak of epidemic diarrhœa which occurred in Manchester in 1894 (*vide infra*), and has also examined a large number of town and country milks. His conclusion is that :—

" Epidemic diarrhœa of the common type occurring in this country is apparently, in the great majority of instances, the result of infection of food by bacilli belonging to the colon group of bacilli, and which

are present at times in fæcal matter. It appears that this infection of food does not generally lead to serious consequences, unless the infection is massive from the first, or the food is kept for a sufficient length of time, and under conditions of temperature favouring the multiplication of these bacilli.

"Milk, which is the most common cause of epidemic diarrhœa in infants, is usually infected at the farm or (through vessels) in transit. Of the bacilli of the colon group which are capable of rendering the milk infectious, those which do not produce a large amount of acid, and do not coagulate milk, are the most virulent, and are probably the essential cause of epidemic diarrhœa!

"Flügge and Sübert believe that cows' milk contains at certain seasons ærobic spore-bearing bacilli of the subtilis type, which have the power of decomposing and peptonising milk, producing metabolic products, which when swallowed by animals cause diarrhœa, etc., and which may therefore produce diarrhœa in infants."

In preparing this work I gratefully acknowledge the courtesy of the Council of the Royal College of Physicians, Edinburgh, and the assistance and advice of Dr Noel Paton, Director of Research, of Dr W. T. Ritchie, and of the Laboratory assistants. I have also to thank Dr Paton for performing five experiments for me.

SPIRILLOSIS OF CATTLE.

By A. THEILER, Bacteriologist to the Transvaal Government, Pretoria.

DEFINITION.

Nocard and Leclainche, in their book *Les maladies microbiennes*, introduced the name "spirillosis" for a disease in geese, due to the presence of a spirillum in the blood of the sick birds (Spirochæte anserina). This spirillum was discovered by Sakharoff in the Trans-Caucus, and was the first pathogenic microbe of the genus spirillum found in animals. A disease in men also due to a spirillum (Spiro-chæte Obermeyeri) has been known for many years under the name of "recurrent fever."

In 1902 I noticed a certain spirillum in cattle, but at that time I did not attach much importance to this microbe. Since then I met with the same parasite in several instances, and always in company with definite pathological features.

At the beginning of last year Laveran, to whom I sent several preparations from sick animals, made a communication regarding the subject to the Academie des Sciences under the name " sur la spiril lose des Bovides," and called the micro-organism " spirillum Theileri." In September of last year Drs Marchoux and Salimbene published an article on " La spirillose des poules " in the Annals of the Pasteur Institute. This disease was found in fowls in and around the town of Rio de Janeiro, and was of a very deadly character. That is there fore the third disease observed in animals known under the name of spirillosis.

I found the spirillum in cattle belonging to the Bacteriological Laboratory, and in the neighbourhood of Pretoria. Probably the parasite will be met with all over South Africa.

The micro-organism in question is a typical spirillum, and varies considerably in its length. The longest microbes measure from 20 to 30 μ. They are somewhat thinner at both ends, otherwise the thickness is about the same throughout the whole length—viz., about $\frac{1}{4}$ to $\frac{1}{3}$ μ. The corkscrew-like forms are, as a rule, predominant, but there are other forms which are atypical in appearance and also shorter. For instance, the parasite may show itself as a simple curved line without any spiral curves, representing sometimes the shape of S ; it may also be completely doubled up, both ends may meet and take the shape of a ring, or both ends may cross each other and also form a loop. These loops may be of different shapes and sizes. Double loops may also be found, like the figure 8. It is somewhat difficult to describe all the different forms, but they can be easily imagined, considering that the spirillum is a very flexible and agile parasite.

In preparations made by placing a cover-glass on a drop of fresh blood the micro-organisms are easily detected. Some of the red corpuscles show a slight irregular motion, and when closely watched it may be noticed that this disturbance may proceed in a certain direction, or again whirl round in the same place. When examined under a high power ($\frac{1}{12}$th inch obj.) the agile spirillum is usually seen attached to one or more red corpuscles. Sometimes it is curled all round a red or white corpuscle. As soon as the organism becomes free and begins to travel through a clear space, a characteristic un-dulating movement is visible, which continues until it finally attaches itself to some other blood corpuscles. This motion may be noticed for some time ; it then becomes relaxed, and finally slackens down completely. I have observed the movements in preparations which were twenty-four hours old.

Staining of the organism may be obtained with any of the aniline dyes used in bacteriology, viz., methylene-blue, fuchsin, thionin. Good preparations were obtained with Laveran's modification of Romanowsky's stain, and also with Azur II. In using the last two mentioned methods no chromatic body could be traced, such as is present in parasites belonging to the protozoa.

Cultivations on the usual artificial media were repeatedly tried, but always with negative results. The same has been found to be the case with all the above-mentioned different spirilla found in men and birds.

The number of parasites present is subject to much variation. For instance, the blood of one animal may show but very few organisms. After tracing the disturbance in the living blood, it took me over an hour to find the parasite in the stained preparations. In other cases they were very frequent, and as many as half-a-dozen and more were seen in the microscopic field. Sometimes two or more were found attached. The number of parasites may also vary in the same animal at different intervals. I have noticed that they may disappear com-pletely overnight, never to be observed again. In one case they disappeared, and only recurred after an interval of about ten days. This suggests a certain similarity to the recurrent fever in man, where

the spirilla are noticed at intervals. Sufficient data are not yet at my disposal to maintain absolutely that the new spirillum produces recurrent fever.

CLINICAL CASES.

CASE I.—1st June 1902. Africander ox, full grown. It arrived a few weeks ago with a herd of cattle from the Orange River Colony. Ordinary red-water broke out amongst the herd and nearly exterminated it. The ox in question was noticed to be very ill. When under my examination it was in a state of collapse. The temperature was 99·2° F. Slight diarrhœa was present. The number of red corpuscles present were 1,900,000 per cmm.

Post-mortem was made soon after death. The cadaver was very much emaciated. There was a general jaundiced condition present, and the fat was replaced by a flabby gelatinous tissue. The blood was very pale. All the lymphatic glands were enlarged and watery. The lungs were found in a state of œdema. The heart was found in diastole, and contained a red clot. The flesh of the heart muscle was flabby and soft. There were no hæmorrhages present, either on endocardium or epicardium. The spleen was very much enlarged and the pulp was soft. The liver was completely icteric and soft. The bile ducts were filled with brown bile. The gall bladder contained the same liquid. The kidneys were normal, and the surrounding connective tissue was infiltrated with a reddish-yellow liquid. The urine was clear, its reaction alkaline, the specific gravity 1015. Esbach's test gave a precipitation of a considerable quantity of albumen. The mesentery was infiltrated with a serous fluid at its junction with the intestine; the mesenteric glands were much enlarged. The fourth stomach was empty; the mucosa was slightly hyperæmic. The mucous membrane of the small intestines was bile-stained, congested, and slightly swollen. The mucosa of the colon, cæcum, and rectum was of a slate colour.

Microscopical examination of the blood revealed the presence of a spirillum. There were numerous basophile erythrocytes. A careful search was made for piroplasma bigeminum, but preparations made with blood from the heart, spleen, liver, and kidneys proved their absence.

CASE II.—8th January 1903. Cape ox, No. 14. This animal was injected intrajugularly with 50 cc., and subcutaneously with 100 cc., of the blood of an ox suffering from East Coast fever. The blood had been sent from Belfast, on the high veldt. The animal was then kept isolated. On the 4th of January 1903 the temperature rose to 105·8° F., and during the following days the ox appeared to be ill, lying down constantly and refusing to eat. When injected it was in a weak state, but lost condition visibly the last few days. On the 7th January 1903 its blood was examined, and the spirillum was found to be present in fair numbers. The ox died early on the morning of the 8th January 1903.

Post-mortem was made about three hours later. The cadaver was very poor, and the flesh was pale. The subcutaneous tissue of the shoulder, breast, and abdomen was infiltrated with a gelatinous liquid. The blood was pale. In the pleural cavity there was a good quantity of yellow liquid. The lungs were œdematous. The pericardium was

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thickened by infiltration with a gelatinous liquid, and there was an increased quantity of serous fluid in the pericardium. The base of the heart and the sulci transversales and longitudinales were also thickened with a similar gelatinous-looking fluid. There was a coat of white fibrous-like tissue on the epicardium, giving the surface a white patchy appearance. The myocardium was very pale; the endocardium was normal. There was a well-formed clot in the ventricles. The spleen was slightly congested and soft, weighing 2½ lbs. The liver weighed 7 lbs. 12 ozs.; it was hard and the section had a glossy appearance. The gall bladder was contracted, and contained viscid bile of a brown colour. The kidneys were studded with white spots about the size of a pin's head. The urine was of normal colour, and its specific gravity was 1012. With Esbach's test a slight precipitation was formed. The omentum was dotted with numerous hæmorrhages, averaging about the diameter of a pea, containing a small clot of coagulated blood; in other parts it was infiltrated with liquid. The mesentery, the serosa of the intestines, the connective tissue of the kidneys and of the pelvis, were also infiltrated with the same liquid; there was an enormous quantity of serous fluid in the peritoneal cavity. The first two stomachs were normal, the third contained soft food. The mucosa of the fourth stomach was very pale. Duodenum, jejunum, and ileum were slightly congested; at some places a superficial necrosis of the mucosa was present. The mucosa of the colon and cæcum was somewhat thickened. The contents of the bowels were soft. All lymphatic glands were enlarged. Smears were made of the different organs. There was a well-pronounced poikylocytosis, basophile granulations were frequent, and the bacillary piroplasma of East Coast fever was present.

Defibrinated blood of this ox was injected into other animals to which I will refer later.

CASE III.—4th February 1903. Cape ox No. 355. This animal was used for the production of rinderpest serum, and had received several injections of rinderpest blood. To-day it was noticed to be very ill. An examination of the blood showed the presence of the spirillum; there were basophile red corpuscles, and also the piroplasma bigeminum. The animal died in the night of·the 4th to the 5th of February 1903.

Post-mortem was made early on the morning of the 5th. The cadaver was poor, there was a general jaundiced appearance over the whole body, but nothing abnormal in the pleural cavity, and the lungs were also normal. The epicardium was studded with petechiæ. The endocardium was normal. A well-formed clot was found in the ventricles. The liver was yellow; the bile viscid and of a brown colour. The spleen was enormously enlarged and very soft. All lymphatic glands were enlarged. The kidneys were anæmic. The urine was yellow. The mucous membrane of the stomach and the duodenum was of a slate colour; the mucosa of the jejunum was bile-stained. There were a few patchy hæmorrhages in the colon and viscid mucus was found in the rectum.

CASE IV.—12th May 1903. Queensland heifer. This animal belonged to a herd of cattle imported from Queensland. Some time back they had been inoculated for ordinary red-water. Part of the cattle were running in the neighbourhood of Pretoria, where some died.

On *post-mortem* the lesions of common red-water, but without red urine, were generally noticed. The examination of blood proved in every instance the absence of piroplasma bigeminum, but the presence of basophile granulations.

On this particular date two heifers died. One of them showed on *post-mortem* the typical red urine, and microscopical examination proved the presence of piroplosma bigeminum.

The *post-mortem* of the second heifer, which had died half-an-hour before my arrival, was as follows: The cadaver was in a poor condition. Rigor mortis was not yet present. The blood was still liquid and had a brownish hue; when put in a pipette the serum which separated had a greenish appearance. The connective tissue and the serous membranes had a yellow tinge. Nothing particular was found in the organs of the pleural cavity. The heart was in diastole and filled with liquid blood. The liver was enlarged, deep yellow, and very friable. The gall bladder was half full, with a thick brown bile. The spleen was enormously enlarged and soft. The kidneys were very pale, and the hilus was infiltrated with a gelatinous-looking fluid. The urine was normal. The intestines were very pale, but otherwise normal.

Microscopical examination of blood demonstrated the absence of piroplasma bigeminum, the presence of numerous basophile granulations, and a well-pronounced poikylocytosis. The spirillum was found in large numbers.

CASE V.—29th June 1903. Heifer No. 208. Mr Thomas Dale, M.R.C.V.S., Veterinary-Surgeon of the Repatriation Department, brought some smears to-day of a sick heifer, and suggested that the spirillum might be present. An examination of the blood showed the presence of the parasite, together with a few piroplasma bigeminum and many basophile granulations.

We arranged to bring the animal to our Experimental Station, where it arrived on the 2nd July 1903. The heifer was in a poor condition. On the 3rd July 1903 the blood was examined, but with the exception of a few basophile granulations nothing was found. The animal was kept under observation, and the temperature was regularly taken. It kept normal for the next few days, but began to rise on the 16th July 1903. Daily examinations were now made, and on the 18th the spirillum was found to reappear; it was only seen on that particular day, and a high temperature was found for about ten days afterwards. The rise of temperature again occurred after an interval of about a fortnight, but the spirillum was found no longer. The animal then became very weak, and dropsical swellings of the head and the dew-lap set in. Notwithstanding the greatest care, the animal died on the 9th November 1903.

On *post-mortem* nothing was found but a very emaciated condition, and the cause of death was put down to debility.

When the heifer showed the spirillum for the second time, blood was withdrawn and other animals were injected. I will allude later on to these experiments.

CASE VI.—5th October 1903. Cape ox, full grown, No. 41. This ox was under treatment for the production of rinderpest serum. It underwent the simultaneous inoculation on the 17th September 1903, and passed through the usual reaction of rinderpest nicely

after the temperature rose again and a second reaction set in, the fever reaching 107° F. The blood of the animal was then examined, when the presence of a spirillum could be detected. It should be mentioned here that the same blood used as virus for this animal had also been injected into five other oxen, in quantities of 2000 cc. per animal, and that these animals did not react. This blood was free of parasites before it was injected. The ox showed symptoms of a very severe illness, refused to eat, stopped chewing its cud, lying down the greater part of the day. The head and ears drooped, the flanks fell in, and the animal lost condition rapidly. It slightly rallied, however, for a considerable length of time. Already on the 5th October 1903 basophile granulations could be seen in the erythrocytes.

On the 6th October 1903 a very pronounced poikylocytosis was apparent, there were many basophile red corpuscles, and also basophile granulations in megaloblasts and microcytes. The spirillum was still abundant.

In the morning of the 7th October 1903 the temperature had dropped to 99·4° F., and the examination of the blood proved the absence of spirillum. The basophile granulations were still frequent.

The animal began to feed again on the 8th October 1903. There were no longer any spirilla found after this date; the basophile granulated cells also became less in number, and on the 16th October 1903 they had completely disappeared.

On the 5th October, when the spirillum was first noticed, the ox was tapped, and the blood was injected into other animals. Of these experiments I shall speak later.

ANALYSES OF THE CLINICAL CASES.

In three of the six cases (Case II., III., and V.) the spirillum was found together with a piroplasma, viz., the piroplasma of East Coast fever in Case II., and with piroplasma bigeminum in Cases III. and V. Cases I. and IV. were observed in a herd which was attacked by ordinary red-water, and, although these two animals did not show any endoglobular parasites, the morbid lesions found indicated that in all probability they had been suffering from red-water. Case VI. was the only case in which in all probability ordinary red-water could be excluded, although the presence of basophile granulations seen on the day the spirillum was first found might perhaps indicate a previous slight red-water infection.

Out of the six cases, three originated spontaneously, whereas three were subsequent to the injections of defibrinated blood to which the occurrence of the spirillum might be attributed. I propose to exclude Case II. from further consideration, as I am at present unable to comprehend how the animal contracted East Coast fever, since it had never been in contact with an infected area; and, although it was injected with blood of East Coast fever, such blood never produces the disease. I may state here that I have reason to believe that bacillary piroplasms are also found in other diseases than East Coast fever, but I cannot as yet substantiate this statement.

The question now arises, is the spirillosis a disease *sui generis*, or is it a symbiotic disease, or is this micro-organism perhaps only a harmless casual parasite?

This last theory cannot be admitted, since the presence of spirillum alone (Cases V. and VI.) gives rise to a very high fever.

Here I would emphasise that the Pretoria district in which the different animals were observed is a red-water infected area. Cattle not immune against this disease usually contract it when exposed to infection ; on the other hand, cattle which are immune against red-water still retain in their system the piroplasma bigeminum, which under ordinary circumstances cannot be seen with the microscope, but is easily traced when such blood is injected into a healthy susceptible animal. Now, when an immune animal happens to be exposed to any adverse conditions in which the vitality becomes reduced, then the piroplasma bigeminum may reoccur in the blood stream and red-water in some modification may arise.

I have seen piroplasmosis complicating rinderpest, trypanasomiasis, pleuro-pneumonia, and to a large extent East Coast fever.

This last malady, which is due to the presence of a bacillary piroplasma, is very often accompanied with the ordinary piroplasma, indeed so often that the first observers confused the two diseases under the name of virulent red-water. It is beyond doubt that red-water together with East Coast fever is due to the recurrence of the piroplasma bigeminum, which is already in the immune animal.

This leads me to suggest that with regard to spirillosis similar conditions exist, namely, the presence of spirillum causes the reappearance of piroplasma bigeminum ; but the evidence seems to indicate that it is rather the red-water parasite which makes possible the medium for the development of the spirillum. Further observations will be necessary to finally settle this question. This fact is indisputable, that spirillosis is as a rule observed in company with red-water.

The *post-mortem* lesions all correspond with ordinary red-water, and it was only in the first two cases that the collection of serous fluid in the peritoneal cavity was noticed, which, however, is a rather abnormal state of red-water.

Comparing the pathological lesions of the cattle spirillosis with the other diseases which, however, are not caused by a mixed infection, but are due to a certain spirillum, we find in lethal cases of recurrent fever in men an enlarged spleen, from four to six times its normal size, complicated with icterus ; and in spirillosis of geese the spleen is softened ; whereas in spirillosis in fowls it is retracted.

In South Africa spirillosis is not at all common. It may be more frequent than is generally supposed, but an accurate diagnosis is only possible by microscopical examination. The uncertainty of the symptoms makes it difficult to arrive at a satisfactory clinical diagnosis.

EXPERIMENTAL INVESTIGATIONS.

I have already mentioned that various animals were injected with blood which contained the spirillum. With reference to Case II., three Texas calves and one Cape ox were injected with 100 cc., 50 cc., and 10 cc. (two animals, respectively, of defibrinated blood directly after it was withdrawn. At the same time one horse was injected with 100 cc. and a second one with 10 cc. One sheep, one goat, and one dog also received 10 cc. each of the same blood ; two rabbits were

injected with 5 cc. into the peritoneum, and two other rabbits under the skin. One rat received about 1 cc. under the skin of the root of the tail.

CASE V.—The following injections were made on the 13th July 1903, that was at the time of the second reaction :—one horse received 20 cc. defibrinated blood subcutaneously, one calf 50 cc. intrajugularly and another 50 cc. subcutaneously, one goat 20 cc. intrajugularly and a second one the same quantity subcutaneously ; two sheep were treated in the same way ; two rabbits were injected with 5 cc. subcutaneously, and one guinea-pig with 3 cc. subcutaneously.

CASE VI.—5th October 1903. The following animals were injected with blood freshly drawn and not defibrinated. Three calves with 50 cc. intrajugularly, one calf with 50 cc. intraperitoneally, one calf with 50 cc. subcutaneously, two sheep with 20 cc. intrajugularly, and one horse with 20 cc. intrajugularly.   '

The result was that none of the injected animals ever showed the slightest reaction, nor the presence of the spirillum in the blood during the subsequent days they were under observation.

This fact does not correspond with the experimental results obtained in the above-mentioned diseases due to spirillum in men, geese, and fowls. The recurrent fever of man is inoculable into the ape. Geese and fowl spirilla are easily transmitted by an injection of blood into susceptible animals of the same species.

We therefore arrive at an unusual phenomenon, which makes it hard to understand how the spirillum comes into the blood of cattle.

TRANSMISSION OF THE SPIRILLUM.

It is through analogy with other diseases that I have tried to explain how the cattle spirillum may be disseminated. For a long time it has been thought that recurrent fever in men is transmitted by acanthia lectularia, it having been found that the spirillum obermeyeri may live over a hundred days in the digestive organs of this insect. Marchoux and Salimbeni have proved beyond doubt that the fowl spirillum is transmitted by a species of argas. Infected ticks conveyed the disease even after five months. But in all these instances we have in all probability to do with a direct inoculation. Argas is a tick which leaves its host after it has had a feed to return again for the same purpose, the night being the time it attacks its prey. Both diseases being easily inoculable, there exists the likelihood of a simple inoculation with infected blood.

The cattle spirillum is, however, as has been demonstrated, not inoculable, and therefore a direct inoculation cannot be considered. On the other hand, ticks which infect South African cattle do not leave the animals unless they are replete, and they do not return again in the same stage, but after they have undergone a moult.

The several ticks behave differently.

Should the cattle spirillum be transmitted by ticks, it would then require to pass through intermediate stages in the life cycle of the tick. We have a striking illustration that such may be the case in the East Coast fever. This disease, due to the bacillary piroplasma, is not inoculable, and yet the brown tick (rhipicephalus appendiculatus) according to my experience carries it as a nympha after it has been

feeding as a larva, and as an adult after it has been feeding as a nympha on sick animals.

Experiments will therefore have to be made in this direction. Of course the spirillum may also enter the system as a secondary infection from the intestines, but at this moment there is no analogy with another spirillum to give rise to such a conclusion.

I here may mention that I have also found the spirillum in the blood of a horse and a sheep. The organisms were present during a fever reaction. Both animals recovered.

ACUTE DISEASE AND PERFORATIONS OF THE ALIMENTARY TRACT, WITH NOTES UPON PERITONITIS.[1]

By EDRED M. CORNER, M.A., M.B., B.Sc. (Cantab), F.R.C.S (Eng.) ; Surgeon to Out-patients, St. Thomas' Hospital ; Assistant Surgeon to the Hospital for Sick Children, Great Ormond Street.

IT is my desire to bring home to my hearers the value of a careful examination of the records of veterinary surgery and pathology. A most fascinating study is afforded in this way, and one which will do much to aid and extend the experimental researches. Acute infective necrosis occurs in animals in the shape of gangrene of tracts and perforations of the alimentary canal. Unfortunately, as yet veterinary pathology is not in so advanced a state as is human pathology, and the records and descriptions are consequently sometimes difficult to understand. Speaking generally, the diseases of animals are not as yet well worked out. But the future will doubtless furnish far greater stores than the somewhat meagre accounts now available.

Thrombosis of a Mesenteric Vein.

I found the record of one case of mesenteric thrombosis in animals described by Connochie in the *Veterinary Journal* of 1898, XLVI., p. 324. The title of the paper is "Vomition in the Horse due to Mesenteric Embolism," and is very misleading. The subject was a valuable hackney filly, rising three years old, which was in good condition. For two days she was noticed to be eating sparely ; on the third day she was found lying down and rolling over and over, and on the fourth day she died. At the *post-mortem* examination an ante-mortem clot was found in the anterior mesenteric vein, with resulting gangrene of the "intestines." The case in reality seemed more acute than many of those of venous thrombosis occurring in man.

Suppurative Typhlitis.

A case of typhlitis such as must correspond with our appendicitis was also found, and is reproduced in this section. A horse was

suddenly seized with violent colic, and died in "some" days. At the autopsy "a quantity of reddish serum was found in the abdominal cavity, and flakes of lymph upon the intestines. On removing the cæcum there was found at the place of attachment an induration from one to two centimetres thick, and in this an abscess the size of a 'nut' which contained grey, thick pus. Along the whole extent of the crook of the cæcum the wall of the gut was thickened. The mucous membrane was very much inflamed, deep red in colour, and the epithelium came away in flakes leaving a blackish corium."

The importance of this case in corresponding to appendicitis in man has been remarked on in that section of the second Erasmus Wilson Lecture, 1904.

Perforations of the Stomach.

Perforated gastric ulcer in animals is a subject covered with much confusion. Acute abdominal disease in animals seems largely to consist of three kinds, namely, colic, ruptured stomach, and twisted guts. From the descriptions it is not easy always to decide upon the case as to whether the condition was inflammatory or the result of true bursting of the stomach, which does occur in animals. Three genuine cases were found.

Journal of Comparative Medicine, 1897, XVIII., p. 716. A horse died from "colic," but was found to have an irregular zigzag ulceration along the greater curvature of the stomach. There was also another ulcer near the pylorus. In the first ulcer was a perforation about one inch long which had caused peritonitis and death.

John Freeman, F.R.C.V.S., *The Veterinarian*, LXXIV., 1901, p. 108. A Scotch deerhound had been ailing for some time with wasting. At first the diagnosis was that of worms, later pernicious anæmia. *Post-mortem*, a perforated gastric ulcer the size of a half-crown was found. The other viscera were healthy.

Mosser, *Bull. de la Soc. Centrale de Méd. Vétérinaire*, 23rd February 1903. At the *post-mortem* examination of a cow ten to twelve ulcers were found along the greater curvature of the stomach, varying in size from that of a pea to that of a franc piece. One of these had perforated and caused peritonitis and death.

Ruptures of the stomach have been reported in far greater numbers than have true perforations, and occur always along the cardiac end of the greater curvature, being sometimes as much as 18 inches long. Mr Malcolm, F.R.C.V.S., says that it is not an infrequent occurrence, and that it is due to the violent contractions of a relatively small and weak stomach which cannot relieve itself by vomiting. In a certain number of these ruptured stomachs there is evidence of previous pathological change. For instance, in the case of Littlewood's, *Veterinarian*, 1894, XXXIV., p. 291, the walls were thickened and softened by parasites.

Perforation of the Small Bowel.

Professor Penberthy, in the *Journal of Comparative Pathology*, 1896, p. 25, says that in enteritis "mortification may occur in eight to ten hours." Such rapidity demands the assumption of the action of micro-organisms.

Perforations of the small bowel seem more common than they are

in any other situation. In the *Journal of Comparative Medicine,* H. H. Dell records a case of three perforations in the ileum, associated with the presence of Tænia Saginata. The subject was a Gordon Setter bitch. He quotes Cadeac, *Rev. Vétérinaire,* 1888, as reporting a case of perforation of the duodenum of a terrier, due to "Tænia." Lahoque, *Receuil Vétérinaire,* 1888, describes three perforations in the small bowel of a dog, due to Tænia serrata.

In the *Journal of Comparative Medicine,* 1899, XX., p. 379, it is reported that a seven-year-old bay mare died, and at the *post-mortem* a perforation of the ileum, half an inch in diameter, was found, and general peritonitis. The ulcer was solitary, and there was no obvious cause.

Perforations of the Large Bowel.

Captain Martin, in the *Veterinary Journal,* 1903, N.S., VII., Pt. 41, p. 273, gives a most interesting account of a unique case. A chestnut mare was found to have an intense imflammation of the cæcum, or typhlitis, with perforation, and through that perforation a number of coils of small intestine had passed into the cæcum and become strangulated.

Davis, *Veterinary Journal,* 1897, XLIV., p. 170. The "floating colon" was found to be greatly inflamed over a patch about 8 inches long, and perforated. A calculus of 2½ lbs. weight was free in the abdominal cavity.

Professor Dewar, *Veterinarian.* A gelding, eight years old, was found, *post-mortem,* to have a "ruptured" colon 10 inches in length. The walls were three to four times as thick as normal.

Neyland and Leblanc, *Veterinarian,* 1896, LXIX., p. 48. *Post-mortem* on a mare who had had "colic." One metre from the anus the gut was ruptured, the "tear" being 10 inches in length, and a large intestinal calculus was in the perforation.

Peritonitis in Animals.

The natural thing to look into to find support for the previously recounted cases was to refer to the records of cases of peritonitis and abdominal abscess. Unfortunately, the origin of these diseases is not always known or looked for. For instance, in the *Veterinary Journal,* 1900, N.S., L., p. 295, a report of a case is given from the *Berlin Theirärzt. Wochenschrift* of a cow which had a large abscess in the peritoneum which discharged per rectum. There was "quite a litre of greyish yellow stinking filthy pus." The sinus closed, and the animal recovered. The origin of the abscess is not known, but had such an accident occurred in a young human being, there would be little or no doubt that the appendix would be at the bottom of the trouble.

The presence of peritonitis, unfortunately, renders the animal unfit for food, and in consequence has not the importance in the veterinary world that it has with us. Brett, *Veterinarian,* 1889, LXII., p. 619, says that peritonitis is not common in cattle, and he gives accounts of three cases which were diagnosed as fæcal impaction. *Post-mortem,* two were called idiopathic peritonitis, and the third was regarded as due to "cold following calving." Littlewood, in the *Veterinarian,* 1890, LXIII., p. 204-207, describes an epidemic of peritonitis which occurred among the horses of the Egyptian Police Reserve, in which

about fourteen horses died of peritonitis, for which no cause was demonstrated. Poisoning was excluded. All cases were acute, and the only definite thing noticed was that the inflammation was mostly in the pelvis and "double colon." Wohmuth, *Veterinarian*, 1901, LXXIV., p. 29-31, records three cases—one being due to a nail in the rumen; another to an abscess in the wall of the rumen; and the third consequent on parturition. The other records merely illustrate its occurrence after operation or parturition.

Looking through these cases, veterinary surgery seems in the same state as that in which the surgery of mankind was some twenty years ago. The idiopathic peritonitis of the latter has turned out to be mainly due to the disease of the appendix, which our predecessors had overlooked. It remains with the future to see if those of the animal will find a similar explanation. In discussing the pathology and physiology of the cæcal region, it was shown that there was every reason to expect inflammation in this place, and also the presence of lymphoid tissue. Beyond the influence of diet and position, there seemed to be no particular reason why this region should be very often affected in the biped and escape in the quadruped. It is to be expected that the disease, when closer attention is paid to it, will be far more frequent in the latter than it appears to be. Veterinary surgeons have not, as yet, recognised anything of the kind. It has also been shown that the collection of lymphoid tissue in the caput coli of animals is the analogue of the appendix of man. And, therefore, it should also have some analogous diseases. It must have a somewhat similar rôle with regard to the action and growth of micro-organisms. And it will, therefore, be liable to like inflammatory changes. These have not been fully recognised, but I have brought forward a case of abscess of the wall of the cæcum, examples of local-ised intraperitoneal abscess near it and in the pelvis, peritonitis limited to and especially marked in the same region. And it is suggested that these have an origin analogous to what they have in man—*i.e.*, the appendix, in the lymphoid tissue of the cæcum.

Professor Clifford Allbutt once wrote to me: "I warmly welcome any attempt to get away from anthropocentric medicine, to escape from the Ptolemaic phase into the cosmic."

Let this sentence close the remarks that I have made upon the subject.

THE COLOUR-REACTION OF ANTHRAX BLOOD WITH METHYLENE-BLUE: A QUESTION OF PRIORITY OF PUBLICATION.

By J. M'FADYEAN, Royal Veterinary College, London.

IN the preceding volume of this *Journal* (p. 35), I published an article entitled "A peculiar reaction of the blood of animals dead of anthrax," in which I called attention to the great value, for purposes of diagnosis, of treating incompletely fixed films of the suspected blood with aqueous solution of methylene-blue. In describing the reaction which is thus brought out, I said that, so far as I was aware, it had not previously been described. In a letter which I have recently received from Professor Heim, of the University of Erlangen, he has

called my attention to the fact that two years previously he had described the same reaction in an article which appeared in the *Archiv für Hygiene* (Vol. XL., p. 55). I therefore take the earliest opportunity to express my regret that this article had escaped my notice, and that I consequently failed to cite it and to give its author the credit to which he is entitled. It may perhaps be regarded as an extenuation of the oversight that the article was simply entitled "Zur Milzbrandinfektion," and was therefore not likely to attract the attention of anyone searching recent literature on the staining of the anthrax bacillus. Indeed, so far as regards its bearing on the question of diagnosis, it appears to have almost entirely escaped notice even in the country in which it was published, and a perusal of the article shows that this is not surprising, apart altogether from the somewhat vague heading under which it appeared.

In the article in question Professor Heim points out that as long ago as 1892 Weichselbaum had mentioned the violet or rose-colour which the envelope of the anthrax bacillus sometimes assumes in preparations stained with methylene-blue. Professor Heim rediscovered this fact, and, what was more important, he also found that preparations of anthrax blood when stained with methylene-blue often showed a more or less abundant rose-coloured amorphous material between the bacilli.

As stated in the second note which I published on the subject in the preceding number of this *Journal* (p. 360), the fact that films of anthrax blood yield a peculiar reaction with methylene-blue has been known to me for a number of years, and a reference to it, and to its value in the diagnosis of the disease, was made in an article which appeared in the *Journal* in 1899 (Vol. XII. p. 177). I confess, however, that at that time I had not accurately realised what were the conditions necessary to obtain the characteristic reaction, or to bring it out with such distinctness as make it visible to the naked eye.

Professor Heim attaches little or no importance to the precise directions as to the method of treating the film which were given in my note of March last. Indeed, he expresses the expectation that I have already discovered that I was mistaken with regard to this point. Such, however, is far from being the case, for further experience has only served to confirm the view that the condition absolutely essential to the success of this method of staining is that the film must not be completely fixed. All methods of treating the film which effect fixation of the red corpuscles prevent the reaction, apparently because they also fix the envelopes or capsules of the bacilli. As previously stated, no trace of the violet reaction can be obtained if the film has been fixed with sublimate, formalin, or osmic acid solutions. Immersion for a few hours in absolute alcohol similarly abolishes the reaction, as does also prolonged dessication of the film even at temperatures far below 100 C. As this is a matter readily put to the test of experiment, it may confidently be predicted that all difference of opinion with regard to it will soon disappear.

Coming in the next place to the question of the origin and significance of the amorphous material which exhibits the violet tint, I find that Professor Heim says (loc. cit., p. 58): " In the degenerated bacilli the rose-coloured part (Rosateil) is at first swollen, and then it ceases to be sharply defined, passing diffusely over into the surround-

ing space, and appearing as if it had flowed out ; frequently it is found
only in the fluid, so that the field of the microscope is thickly covered
with amorphous fragments (schollenartigen Gebilden), which have often
retained the bacillary form." With all this I agree, save that I still
cannot assent to the interpretation of the picture which is implied in
the word " degenerated." The material is undoubtedly derived from
the so-called envelope or capsule which anthrax bacilli formed in the
animal body have long been known to possess, but there does not
appear to be any good ground for regarding the formation of this
capsule as evidence of a degeneration on the part of the bacilli. This
capsule is invariably present in the bacilli found in the fresh blood
and organs, though it is perhaps thicker in the blood rods than in
those formed elsewhere in the body. Moreover, it is present in
connection with the bacilli that must have been actively multiplying
at the moment of death.

The distorted appearance of the capsules is in my opinion an
artefact, produced by the addition of the watery stain to the imper-
fectly fixed film. The proof of the correctness of that view is easily
obtained. All that is necessary is to examine a dry film made with
blood from on animal just dead from anthrax, or to fix such a film
completely by dry heat, alcohol, or sublimate or osmic acid solution,
and then to stain it by the method of Olt or Johne. It will then be
found that the appearances above described as evidence of degenera-
tion are entirely lacking, the bacilli being provided with capsules that
are sharply defined outwardly, and free from those irregularities of
contour supposed by Professor Heim to indicate liquefaction. On
the other hand, if another film of the same blood be prepared, and
stained with methylene-blue after incomplete fixation by heat or
immersion for a few minutes in alcohol, it will be found that a great
many of the bacilli show no capsule at all, while others are partially
or completely surrounded by a reddish-purple material, and the field
between the bacilli is everywhere occupied by larger or smaller
amorphous particles which exhibit the same tint.

The true explanation of the peculiar reaction of fresh anthrax
blood with methylene-blue thus appears to be (1) that when the
unfixed bacilli are treated with water the outer part of their bodies
(the so-called capsule) swells up and in large measure disintegrates,
and (2) that the swollen capsules and the particles into which they
disintegrate behave in a metachromatic manner with regard to
aqueous solution of methylene-blue.

This explanation applies also to those cases in which the stained
film shows few or no anthrax bacilli, but neverthless a large number
of purple-stained amorphous granules scattered throughout the field.
These granules are the result not of the degeneration of the bacilli,
but of their death and disintegration, and the reaction is obtainable
for a considerable period after the death of the animal, and is there-
fore valuable for diagnosis, because the capsules are the most resistant
part of the bacilli, and the last to disappear when putrefaction sets in.

In conclusion, it ought to be observed that throughout this note
the word methylene-blue has been used merely for the sake of
brevity, for, as already explained (see this *Journal,* Vol. XVI., p. 360),
there is reason to suppose that the reaction is not obtainable with a
solution of the pure dye.

Reviews.

The Principles and Practice of Veterinary Surgery. By William Williams, F.R.C.V.S.. F.R.S.E., etc. Tenth edition. Revised by W. Owen Williams, F.R.C.V.S., F.R.S.E. London: Bailliere, Tindall & Cox, 1904.

THE tenth edition of a work so well known to the members of the veterinary profession as this is hardly calls for a lengthened notice. It is true that the death of the distinguished author of the work since the publication of the previous edition has thrown on his son, Professor Owen Williams, the respon-sibility of bringing the matter up to date, but the revision to which the text has been subjected has been of the lightest possible character. It may truly be said that the present edition possesses all the merits, but unfortunately also all the defects, of its predecessors. Among its merits may be reckoned the fact that it deals with the surgery of the domesticated animals in a practical manner, and within such reasonable compass as to make the book a convenient one for the use of veterinary students. Its greatest defects are on the scientific side. For the most part the exposition of the pathological processes which are of chief interest to the surgeon is altogether out of date, and reads like excerpts from some human text-books of half a century ago. This is very notably the case in the account which is given of inflammation. It is almost melancholy to find, in a book published in 1904, the view that the formation of pus is ever a healthy process, and to discover that the account which is given of the source and structure of pus corpuscles absolutely ignores everything which has been added to our knowledge of these matters during the last twenty years. It is equally lamentable to find that this edition, like all the preceding ones, contains a long extract from the writings of the late Professor Spence on the pathology of strumous disease of the joints in man, followed by a hint for the benefit of human pathologists that further researches may show them that so-called strumous arthritis is in reality due to altered innervation consequent upon some constitutional taint! It is much to be feared that if this and some other passages should meet the eye of human pathologists and surgeons it will give them a very bad impression of the present state of pathological knowledge among veterinary surgeons in this country.

These, of course, are old errors, for which the editor of the present edition has no responsibility, except that he decided that it was well to allow them to stand. Unfortunately, however, there are other errors which point to real carelessness in revision, for the feelings which prompted a tender hand in dealing with the opinions expressed by the original author need not have prevented the correction of mis-spells and errors of that kind. We think it right to point out a few of these mistakes in order that they may be corrected in the next edition. On page 43 the microbe responsible for equine strangles is said to be the "streptococcus pyogenis," which, by the way, is a mis-statement of fact as well as a mis-spell. On page 450 the central part of an actinomyces colony is said to be "homogeneous," and its outer part like the "capitalum" of a daisy. On page 599 epizoötic lymphangitis is said to be due to a large, slightly ovoid "microccus." This also, it may be observed, is an error of fact, for, although the exact place of the cryptococcus of Rivolta in the vegetable kingdom is perhaps a little doubtful, it is quite certain that it is not a micrococcus.

Points of the Horse. A treatise on the conformation, movements, breeds, and evolution of the horse. By M. Horace Hayes, F.R.C.V.S. Third edition. London : Hurst & Blackett, Ltd., 1904.

THE appearance of a third edition of this work within a few years indicates that the author has "secured the market." It is a result to which he was well entitled from the quality of the book, and the present issue of it is even superior to the last. It exceeds the second edition by no less than 400 pages, part of which is accounted for by 279 new illustrations, and the remainder by important additions and amplifications of the text. The bulk of the new matter has been introduced into the chapters dealing with the various breeds of horses, British and foreign, and a large proportion of the new illustrations relate to the same subject. This part of the work is so full that it is doubtful whether any breed in existence, either at home or abroad, has escaped attention. The figures are almost entirely reproductions of photographs, and with scarcely an exception they are of excellent quality, while the publishers' share of the work generally is beyond praise.

CLINICAL ARTICLES.

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A CASE OF ŒSOPHAGEAL POUCH IN A HORSE.

By J. W. COLEMAN, M.R.C.V.S., Swindon.

Subject.—An aged thoroughbred, purchased by the owner about six weeks before I was consulted.

History.—The day following the purchase the groom noticed that the horse did not eat its food naturally, and that after partaking of some he would continue to champ for a considerable time, and would then have a severe fit of coughing and vomit a considerable portion of the ingesta through his mouth and nostrils. In the channel of the neck, just outside the thorax, he had noticed a movement resembling "a snake attempting to escape up the throat," and suggested that that was what it was, the horse, he thought, having drunk some bad water with a young snake in it !

Symptoms.—(A fortnight before death.) Weird anxious look, staring coat, emaciated condition, slightly accelerated breathing, ingesta exuding from mouth and nostrils, slight distension and regurgitation in œsophagus at lower part of the middle third of the neck, visible mucous membranes congested, pulse 50, temperature 103° F.

Diagnosis.—Partial obstruction of œsophagus (thoracic portion), either by pressure of a tumour, constriction, or rupture of its walls.

Prognosis.—Unsatisfactory ; advised slaughter.

Treatment.—The groom had given the animal a physic ball about three weeks before I saw the case, but it never acted in the least.

I gave the patient a bottle of cold water, which distressed him very much, and induced a fit of coughing, after which he vomited the major portion of the liquid. I then threw the animal, and very carefully passed the probang. I found some difficulty when it reached the cardiac

orifice to get it to enter the stomach, but ultimately succeeded, without using force. When the animal rose he appeared considerably relieved, and I almost hoped I had removed the obstruction. The animal began eating grass, and when the coughing and vomiting recurred I attributed this to the irritated condition of the œsophagus, and ordered a pint of linseed oil to be given. The foregoing symptoms, however, persisted till the end, but were perhaps not quite so intense.

On the 6th October last the animal got down and was unable to rise, so I slaughtered him.

Autopsy.—Pharynx and cervical portions of œsophagus filled (but not distended) with ingesta. Thoracic portion distended and filled with ingesta, and a soft tumour-like swelling of the œsophagus extending from the diaphragm forwards into the thorax for about 8 or 10

inches, and being about 10 to 12 inches in circumference. Upon removal of the gullet from the thorax I found this swelling to be a pouch of the mucous coat herniated through a longitudinal tear of the muscular coat of the tube.

The defect or tear in the muscular coat had rounded edges, and measured about 10 inches in length. It was situated on the upper surface of the tube. Above the pouch or sac the gullet was cylindrically dilated with pultaceous food materials.

After the accompanying photograph had been taken the sac was laid open and emptied of its soft contents, so as to expose the inner surface of the mucous membrane. This appeared quite normal everywhere, and there was no evidence of degeneration in the muscular strata.

CONTAGIOUS DISEASE AFFECTING THE GENITAL ORGANS OF SHEEP.

By G. H. WILLIAMS, M.R.C.V.S., Chippenham.

NOTICING the remarks on this subject by Prof. M'Fadyean and Mr Flook in last number of the *Journal*, it has occurred to me that it might be of interest to record two similar outbreaks which I saw in this district in September last.

In the first outbreak, in a flock of eight ewes and a ram, two ewes and the ram were affected in a benign form, and, though no isolation was carried out, the disease did not spread. There was considerable balanitis and ulceration of the penis. A chinosol and zinc sulphate lotion was used, and the affected parts had healed in about a fortnight.

The other outbreak began about the middle of September last. On the 29th of that month my attention was called to a flock of fifty yearling ewes, and two ram lambs and an old ram running with them. This flock had been bought on the Downs at a fair about three weeks before.

The rams and some forty of the ewes were affected, and showed on all of the genitals the various stages of the lesions described by Mr Flook. One ewe was affected on the nostril. In addition to ulceration and distorsion of the pudenda by the inflammatory swelling, there were in many cases freely bulging granulations of a purplish colour.

Nitrate of silver was used on these granulations, and on the ulcers occasionally, whilst all were treated with the zinc and chinosol.

The old ram had been with another flock of older ewes which had not mixed with the new flock, and to this flock of old ewes my attention was called on 17th October, as a dozen or so had eruptions about the lips and noses, but no genital affection. Another old ram with this lot escaped the disease.

In the young flock, separation of the affected and unaffected was immediately resorted to, a daily examination being made of the apparently free, and freshly developed cases being placed with the affected lot. The rams were also isolated from all ewes.

After three weeks from the last developed case, a new ram was put with the young unaffected ewes, and later on the recovered males and females were placed together. The disease did not recur, and this spring the ewes have dropped normal lambs.

MULTIPLE UTERINE MYOMATA IN A COW.

By A. SPREULL, Junr., M.R.C.V.S., Dundee.

THE subject of this note was a well-nourished cross-bred Ayrshire cow about eight years old, belonging to a farmer. The cow was bought by this farmer in September 1902, and gave birth to a living calf shortly afterwards. From that time up to date of slaughter she milked well and retained her condition. Œstrum appeared at regular intervals, but the cow was never put to the bull. At the same time the abdomen gradually increased in bulk, so that she was by some believed to be pregnant. Three days before slaughter she

became tympanitic whilst grazing on a field of clover. She was treated by the farmer himself, partially recovered, and was sent to the abattoirs to save further trouble and for economic reasons. She was killed on the 8th September last.

On *post-mortem* examination the only organ found to be diseased was the uterus, which was greatly distended and contained fifteen growths, each about the size of a melon, and about a score of much smaller ones. These tumours were well defined, yellowish-white in colour, oval in shape, of the consistence of firm fibrous tissue, and on section showed bundles of fibres crossing each other in very various directions.

Several of the growths appeared in a degenerated condition, being softer than the others, more friable, and having at points in their interior fluid contents of a brownish colour. The whole mass, uterus included, weighed 177 lbs.

One of the smaller tumours was sent to Prof. M'Fadyean, who made a microscopic examination and pronounced it to be a typical non-striped myoma.

A CASE OF EQUINE BACILLARY NECROSIS, WITH METASTATIC LUNG LESIONS.

By W. CAUDWELL, F.R.C.V.S., Chertsey.

Subject.—A valuable brown draught mare, eight years old. I was asked to see her on the 25th June 1903, when I found her very lame with a suppurating wound in the off hind heel, and she had a quick irritable pulse. She had been working on a bad road made of faggots and brick-bats, where she had doubtless received the injury.

Treatment.—Antiseptic fomentations and poultices.

26th June.—Pulse frequent, temperature 105°, respirations rapid, sweating with pain.

27th June.—Symptoms as yesterday, and in addition the limb is much swollen and abscesses are forming around the fetlock. I placed her in slings and applied antiseptic fomentations and poultices.

28th June to 2nd July.—The skin sloughed in patches between the hock and the coronet, and hæmorrhages ensued.

3rd July.—Pulse 120, respiration very rapid, ears cold, and death obviously approaching, so I had her destroyed.

4th July.—*Post-mortem* examination revealed the presence of a large number of abscesses throughout both lungs, especially numerous just beneath the pleura, and about as large in circumference as a five-shilling piece. The skin on the off hind leg from the heel to the hock showed necrotic patches and subcutaneous extravasations and abscesses, and the sheaths of the perforans and perforatus tendons were intensely inflamed and injected.

A microscopical examination of a smear from one of the metastatic abscesses in the lungs showed the presence of what I believed to be necrosis bacilli.

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INTUSSUSCEPTIONS OF THE DYING.

By J. R. BAXTER, M.R.C.V.S., Lechlade, Glos.

MY attention was attracted to the occurrence of this condition by an article in the *Journal of Comparative Pathology*, Vol. XVI., page 154. In that article it was stated that "intussusceptions of the dying" had not been recognised by the veterinary profession ; since then I have had the good fortune to come across a case of this condition while making an autopsy on a cross-bred English terrier pup, which died in a fit shortly after being admitted to the infirmary for treatment for distemper. The condition noted was exactly similar to that described, viz., the intussusception was multiple (four in number, each about eight inches long), very easily reduced, no signs of inflammation or any change whatever, and they were in the small bowel ; in fact, the only respect in which they did not quite accord with description was that they were descending and not ascending.

LUMBAR TUBERCULOSIS, WITH FRACTURE FOLLOWING, IN A HEIFER.

By AINSWORTH WILSON, F.R.C.V.S., Blairgowrie.

I AM induced to publish this case in the belief that the lesions disclosed on *post-mortem* will prove of pathological interest.

The *subject* was a patient of mine a few months ago—a polled Angus (Ballindalloch) heifer, suckling her first calf six weeks old. Kept in a roomy loose box ; all right at night, and found next morning unable to rise. There was no history or sign of an accident, animal in capital condition, and enjoyed previous good health.

Symptoms.—Complete paraplegia ; no control over hind limbs, though able to move the fore part of body ; loss of sensation together with loss of voluntary motor power extending backwards from the posterior lumbar vertebræ ; paralysis of bladder, with retention of urine ; rectum packed with soft fæces, and tail quite flaccid. Constitutional symptoms were absent, respirations, pulse, and temperature being practically normal, and appetite good. Conjunctival mucous membrane a little injected. Manipulation of the loins and hind legs gave negative results.

Diagnosis.—The complete loss of nerve force and the sudden onset of the symptoms made me suspect fracture ; there appeared to be pressure paralysis from some such cause. A fairly definite, though by no means a positive, diagnosis was arrived at by a process of exclusion, other possible causes being eliminated. After emptying the rectum and passing the catheter, the cow was raised in slings for two or three minutes ; she, however, hung helplessly, knuckling over completely at both hind fetlocks. On rectal examination, a firm fixed tumour was detected in the sub-lumbar region, immediately beneath the spinal column.

Prognosis.—Very doubtful ; pressure on the spinal cord, and chances of recovery very poor. The owner, however, decided on treatment, the animal being a valuable pedigree one and a favourite.

Treatment followed the usual lines. Attention to details of nursing, removal of urine and fæces, enemas, the administration of an oleaginous purge, etc.

1st week.—Condition remained unaltered ; she fed well, chewed her cud, and gave a fair amount of milk. Two drachms of nux vomica were given twice daily, the spine and hind legs being hand-rubbed and stimulated.

2nd week.—Involuntary discharge of urine ; by and bye urination and defæcation performed quite freely ; tail a little stiffer, though at no time was the anæsthesia of the hind parts in the least overcome. Half-ounce doses of nux vomica powder and a few doses of the tincture with spt. amm. aromat. were now given, alternated with iodide of potassium, and a biniodide of mercury blister was applied to the spine.

3rd week.—Cow again raised in slings ; increasing weakness, appetite less hearty ; restlessness and grinding of the teeth ; hind quarters wasting very visibly, and, in spite of precautions, some gangrenous ulcers appearing on the teats. The nux vomica, being pushed far enough, was stopped, and a laxative administered. Owner at last consented to have her killed. This was done on the *23rd day*, and a *post-mortem* was made immediately afterwards.

Autopsy.—All the important organs, lymphatic glands, and serous membranes, together with the spinal column, were subjected to a close examination. A few mesenteric glands, four in number, were found enlarged, more or less indurated, and caseous. The largest, the size of a crab apple, showed calcareous changes ; the others, much smaller, showed a few yellow caseous tubercles dotted over the section. Glands of large intestines, mucous membrane, and wall of small intestines healthy. A tuberculous firm fibrous growth was exposed in close connection with the bodies of the last two lumbar vertebræ, in size rather larger than a goose's egg, mainly fibrous in structure, but partly infiltrated with lime salts, and showing several areas of cheesy caseous matter close to the bone. By staining a film (smear) obtained by rubbing a cover-glass over the cut surface, I was able, fortunately, to detect tubercle bacilli.

The lumbar vertebræ were isolated, and a longitudinal section made down the centre with the saw. It was now apparent that a fracture existed of the body and arch of the 5th bone, close to its articulation with the 6th ; these two vertebræ were the seat of a rarefying ostitis, and the following changes were presented : Softening of the bone, dilatation of the spongy spaces and infiltration with yellowish caseous material ; ulceration and partial removal by absorption of the interarticular cartilage, which, as usual, resisted the disintegrating process longer than the osseous vertebræ ; and, lastly, suppuration in connection with the spinal meninges. The absence of any attempt at the formation of new bone, and the close connection of the above-described growth with the diseased spinal bones, were points worthy of note ; there was, in fact, no line of demarcation, the disintegrated deeper part of the growth passing imperceptibly into the softened mass of bone. The compression of the spinal cord to the diameter of a slate pencil in the centre of the diseased area was very striking.

Right kidney appeared a little enlarged, firmer than normal, with greyish slightly-raised areas under the thickened capsule ; pale on section, and raised areas showed as summits of fibrous bands running

through cortex into medulla.   Horizontal sections stained for structure and bacilli respectively showed that the changes were those common to interstitial nephritis, no tuberculous elements being present.   Some extravasation of blood was noted behind the kidneys, together with a swollen œdematous condition of the lumbar lymphatic glands on both sides ; the sacral group and other important glands in the abdomen and chest were, however, to all appearances absolutely healthy.

I regret that the cord was not examined in a fresh state.

Remarks.—The tuberculosis appeared to have been pre-existent to the fracture, and to have acted as its predisposing cause ; in other words, the fracture, with resulting pressure on the spinal cord, was evidently a sequel to the disease in the bone.   It is obvious that a very slight force would suffice to bring about the fracture with the amount of cartilaginous and bony disintegration present.   It is somewhat remarkable that so little tuberculosis existed in other organs, the glands of the mesentery and the vertebræ being alone affected, so far as I could determine with the naked eye.

The somewhat doubtful or suspicious condition of the lumbar glands has been noted above.

In this respect, *i.e.*, the scarcity of tuberculous lesions, I think the case may fairly be regarded as an exceptional one.   I may be pardoned for quoting Professor M'Fadyean in this connection : " Tuberculosis affecting either the osseous system or the articulations is comparatively rare in cattle. . . . The bones most commonly affected are those of the spine, and the disease is generally associated with extensive lesions in the abdominal and thoracic organs and viscera.   Probably it is generally a local tuberculosis, due to infection by the lymphatic system of vessels."[1]

It is always interesting and instructive to trace the course of infection in such cases.   The entire absence of any tubercular foci in the lungs puts auto-infection out of account.   The bacilli appear to have gained access with the food or water, and to have passed straight through the intestinal wall without affecting it ; thence to the mesenteric glands by way of the lacteals, and from there to the lymphatics of the spinal column.

I had lately an opportunity of examining some specimens of tuberculosis of the spine in children, and their resemblance to this case struck me very forcibly.   I was informed that in special cases in the human subject it is quite unusual to find much tuberculous disease in other parts of the body.

[1] "Journ. Comp. Path. and Therap." September 1898.

Abstracts.

PROTECTIVE INOCULATION AGAINST TUBERCULOSIS.

ALTHOUGH Bang's method of combating tuberculosis has been very widely adopted in different countries, great difficulty is experienced in adapting it to the varying circumstances met with, and a reliable process of inoculation, unattended by injury to the subject, would undoubtedly displace it on account of the saving both in time and money. An experimental test of v. Behring's system of protective inoculation is therefore of interest.

Two " protected " animals, viz., a one-and-a-half year-old heifer, No. 14, and a one-and-a-half year-old steer, No. 40, were supplied from von Behring's laboratory at Marburg. Three control animals, No. 1, a six months old calf, No. 2, a one-and-three-quarter years old, and No. 3, a thirteen-months old heifer, were also employed.

The Marburger animals had undergone the following preparatory treatment in von Behring's laboratory :—

No. 14, tested with tuberculin, gave a rise in temperature of 1° C., which continued for several days ; the animal was therefore suspected of tuberculosis. A fortnight later it was intravenously injected with ·001 gramme of dead tubercle bacilli ; five days later with ·5 gramme of spleen emulsion from a guinea-pig infected with very virulent bovine tuberculosis. The animal thereafter suffered for some weeks from cough and fever, and reacted strongly to tuberculin. Ten weeks later it seemed quite to have recovered, and was treated intravenously with ·005 gramme of a culture of human tubercle bacilli ; moderate reaction. A week later it failed to react to a second injection of ·01 gramme.

On 2nd December 1901 it received ·02 gramme of the same culture : short but severe fever reaction.

On 7th December 1901 it received ·025 gramme of the same culture : short reactions ; increase in weight.

On 11th December 1901 it received ·05 gramme of the same culture : short reactions ; increase in weight.

On 14th December 1901 it received ·1 gramme of the same culture : short reactions ; increase in weight.

On 17th December 1901 it received ·2 gramme of the same culture : short reactions ; increase in weight.

On 21st December 1901 it received ·4 gramme of the same culture : short reactions ; increase in weight.

On 8th and 28th January and 17th February 1902, tests with ·2 ccm. of tuberculin produced reaction.

On 27th May 1902, injection of ·25 ccm. tuberculin proved negative.

On 16th July 1902, ·04 gramme of the same human culture produced moderate fever.

Steer No. 40 was tested with tuberculin on 22nd December 1901. Two days later it received intravenously ·025 gramme of a very virulent culture of tubercle bacilli, whereupon severe fever and coughing appeared and continued for over seven weeks. Recovery then occurred.

The heifer was regarded as more immune against tuberculosis than the steer.

The object of the investigation was to test the degree of protection afforded by von Behring's process against deliberate artificial inoculation. On 4th February 1903, the Marburg animals, having recovered from their journey, etc., and being apparently in good health and condition, were injected with ·4 ccm. of Koch's tuberculin. Both showed the typical reaction, the heifer's temperature rising 2·3° C. and the steer's, 2·2° C. This seemed to indicate

that, despite von Behring's treatment, both animals suffered from unhealed tuberculous lesions. Control animal No. 1 was injected at the same time and showed no reaction. The three animals were then placed together under precisely similar conditions, and on the 13th February 1903 were inoculated in the following way :—

From a carefully selected case of bovine tuberculosis a lymphatic gland, which exhibited giant cells and bacilli in moderate numbers, was obtained. ·1 gramme of gland substance was rubbed down with 15 ccm. of sterilised bouillon, and of this material one third, *i.e.*, 5 ccm., was intravenously injected into the left jugular of each animal. Three guinea-pigs, simultaneously inoculated in the peritoneal cavity with 1 ccm. of similar bouillon, died in five, eight, and twelve weeks respectively, with generalised tuberculosis. The material selected gave no growths on artificial media.

The two Marburg animals and the control were now subjected to most careful daily observation. For three weeks nothing of real importance was observed, except for a rise to 41·1° C. in the control animal's temperature on the 5th March 1903. In a week the temperature had again fallen to normal. The temperature of heifer No. 14, on the other hand, always remained below 39° C., and that of steer No. 40 was, on the 3rd March 1903, 39·3° C., on the 6th April 1903, 39·8° C., and on the 16th April 1903, 39·4° C. At no other time did it exceed 39° C. The two Marburg animals remained bright and in good condition, while control animal No. 1 was dull, showed staring coat and diminished appetite. None of the three lost weight in any notable degree.

Nine weeks after infection, when the lesions were thought to have healed, ·5 ccm. of Koch's tuberculin was administered. All three animals reacted strongly, heifer No. 14 showing 1·5° C., steer No. 40, 2·1° C., and control No. 1, 3·9° C. of fever.

It was then resolved not to slaughter control No. 1, but to re-inoculate it together with the Marburg animal with more virulent material and to introduce two more controls into the experiment. The object was, in part, to learn whether control No. 1 was more or less susceptible to a second inoculation as a result of having survived the first. The *post-mortem* might prove less instructive, but the balance of interest lay in re-inoculating.

Control animal No. 2 was a one-and-three-quarter years old, and No. 2, a thirteen months old heifer. Both failed to react to tuberculin.

On the 26th May 1903 each of the five subjects received intravenously 5 ccm. of a solution prepared by rubbing down ·01 gramme of a highly virulent culture of bovine tuberculosis in 100 ccm. of bouillon until the whole formed a uniform emulsion. This 5 ccm. of bouillon therefore contained ·0005 gramme of tubercle bacilli.

One guinea-pig was subcutaneously injected with 5 ccm. and a second intraperitoneally with 2·5 ccm. of similar emulsion. The first died in five, and the second in four weeks, of generalised tuberculosis. The infective material was therefore of marked virulence.

Heifer No. 14 and steer No. 40 showed febrile reactions of 39·6° and 39·7° C. respectively, during the next two days, whilst the temperature of control No. 2 varied as follows : On the sixth day after inoculation, 39·3°, on the thirteenth, 39·3°, on the twenty-seventh, 39·7°, and on the forty-first day, 39·3° C. As regards general condition, appetite, and bodily weight, control No. 2 gave the best results, then followed steer No. 40, whilst heifer No. 14, which had already several times been inoculated with bovine tuberculous material, showed evidence of wasting and was unthrifty in her coat. She had lost weight as follows : On the 4th February 1903, she weighed 269 kgs., on the 26th May 1903, 266·8 kgs., on the 15th July 1903, 245·1 kgs., and on the 1st August 1903, 225·5 kgs.

On the 2nd and 3rd August 1903, heifer No. 14, steer No. 40, and the three

control animals were injected with 5 cc. of 10 per cent. Marburg tuberculin. They showed reactions of 1·2° and 1·6° C. respectively. Control No. 2 showed 2·2° C., and when re-tested on the 23rd September, 2·1° C. Until slaughtered on the 6th October 1903. control No. 2 appeared perfectly healthy, showed no fever, steadily gained in weight, and showed good appetite; occasionally, how- ever, its coat appeared a little unthrifty.

In contrast with control No. 2 ànd steer No. 40, controls Nos. 1 and 3 reacted severely and for a long time to the injection made on the 26th May. In each case the coat appeared dry and staring, the skin tight, the appetite lessened, whilst the animals fell away in condition. These symptoms, however, dimin- ished after the ninth week following infection. During the days immediately following inoculation, the temperature rose as high as 40·8° in one case and 40·3° C. in the other. From the ninth week the temperature in each case remained normal. Injected with tuberculin on the 23rd September, control No. 1 showed a rise in temperature of 1·6° C. and No. 2 of 2·1° C. Though neither of these animals lost weight to any important extent throughout the experiments they showed at irregelar intervals want of appetite, dulness, depres- sion, and an unthrifty and rough state of the coat.

Heifer No. 14 and control No. 2 were only once observed to suffer from cough, and then only for a very short time, but steer No. 40 and controls Nos. 1 and 3 showed slight cough on numerous occasions.

Post-mortem appearances.—Heifer No. 14 was slaughtered in Marburg on the 10th August 1903. The appearances were as follows: Spleen capsule showed fibrous thickening; no tubercle bacilli could be found. The lower portions of the pleura showed flat, prominent, fibrous growths (no tubercle bacilli detected), and at some points pedunculated, calcified, warty excrescences, in which no tubercle bacilli were found. The lower margin of the lung showed four greyish-yellow centres, the size of grains of linseed, lying just under the pleura, and containing a moderate number of tubercle bacilli. Mediastinal glands enlarged and containing yellowish centres the size of a pin's head, in which a few tubercle bacilli were found. Right kidney moderately enlarged; on the margin of the medullary substance a few small centres were just visible; they contained a small number of tubercle bacilli.

Heifer No. 14 had therefore suffered from slight but generalised embolic tuberculosis.

Steer No. 40, having remained in good condition up to the 21st September 1903 and considerably increased in weight, was preserved alive for further observation.

Control No. 1 was slaughtered on the 28th October 1903.

The point of inoculation on the left side of the neck showed a tubercle the size of a grain of linseed, which contained very numerous tubercle bacilli. The left prescapular gland was as large as a hen's egg, but contained no tubercle bacilli. The right prepubic lymphatic gland contained a calcified tubercle as large as a pin's head, showing moderately numerous bacilli. The portai glands showed very numerous calcified tubercles, some as large as a grain of linseed. The serous coat of the spleen was thickened, and the pulp contained some tubercles as large as a grain of linseed (no tubercle bacilli visible). Two of the mesenteric glands showed calcareo-caseous tubercles as large as peas, with scanty tubercle bacilli. In both kidneys were many recent embolic centres varying in size between a pin's head and a grain of linseed, and containing a moderate number of tubercle bacilli. The entire lung showed greyish-yellow, calcareo-caseous embolic centres surrounded with fibrous capsules, some as large as a pea; others of these centres were glassy or greyish-red; they were most numerous under the pleura, but could also be detected in the depth of the parenchyma. The larger, older, calcareo-caseous tubercles were moderately numerous; the pea-sized, glassy or necrotic, more

recent tubercles, very numerous. The bronchial and mediastinal glands were as large as hen's eggs, and contained bean-sized, calcified centres with fibrous capsules. No tubercle bacilli were found in these centres, though more recent ones contained moderate numbers.

Control No. 1 had therefore suffered from generalised embolic tuberculosis.

The centres due to the inoculation of the 13th February 1903 showed a marked tendency towards healing; those due to the injection of the 26th May 1903 were, on the other hand, still fresh.

Control No. 2 was slaughtered on the 6th October 1903. The *post-mortem* appearances were as follows :—

The point of inoculation on the left side of the neck showed a caseated and calcified centre as large as a pigeon's egg, surrounded with a thick capsule of connective tissue and containing numerous tubercle bacilli. The left prescapular lymphatic glands were enlarged, and a space as large as a bean was dotted over with numerous calcified centres as large as pins' heads containing a few bacilli. The right pleura showed several greyish-red villous granulations which were partially adherent to similar granulations on the corresponding portion of the pleura covering the lung (tubercle bacilli not detected). The right posterior margin of the lung showed three calcified tubercles as large as vetch seeds and containing scanty bacilli. The posterior mediastinal glands showed several calcified centres the size of pins' heads (no tubercle bacilli detected).

Control No. 2 had therefore suffered from slight tuberculosis of the point of inoculation, and of the neighbouring lymphatic glands as well as of the thoracic cavity.

The disease showed a distinct tendency towards recovery (marked capsule-formation, marked calcification, sparing number of tubercle bacilli, slight tendency to spread, no generalisation).

Control No. 3 was slaughtered on the 13th October 1903.

On the left side of the neck were three closely-placed chestnut-sized centres containing pus and numerous tubercle bacilli, and surrounded with thick capsules of connective tissue. The left prescapular glands contained two caseated and calcified points as large as grains of linseed, with a few tubercle bacilli. In the right axillary glands were two very small calcified centres containing a very few tubercle bacilli. In the left inguinal gland was a very small calcified nodule with very few tubercle bacilli. In the right inguinal gland were two calcified nodules as large as pins' heads, with very few tubercle bacilli.

Control No. 3 had therefore suffered from a tuberculous abscess at the point of inoculation, together with trifling tuberculosis of the prescapular glands and of the right and left inguinal glands. All the internal organs and all the serous membranes were free of tuberculosis.

The disease showed an unquestionable tendency towards recovery—formation of thick capsules, calcification, number of tubercle bacilli trifling, tendency to extend slight (Schlegel, *Berliner Tierärzt. Woch.*, 1903, p. 745).

PATHOLOGY OF FORAGE POISONING OR SO-CALLED EPIZOOTIC CEREBRO-SPINAL MENINGITIS OF HORSES.

EXPERIMENTS and observations have led Dr Pearson to propose the name "forage poisoning" for this disease, a name which he regards as more in accordance with the facts as we know them at present. The term "cerebro-spinal-meningitis" is not justified by the clinical history nor by *post-mortem* findings. The actual pathogenic agent has not yet been discovered, though a toxic mould or fungus is supposed to be the cause.

Gross examination usually shows hyperæmia of the brain and cord and their meninges, with increase of fluid in the sub-arachnoid spaces and ventricles. No micro-organism has been discovered in this fluid by cultural methods.

In mild attacks of the disease there is loss of control over the limbs and tail, loss of appetite, and difficulty in swallowing, which is well marked in severe cases. Stupor, apathy, and extreme muscular weakness or actual paralysis exist; contraction of the muscles of the neck, back, and loins, with more or less opisthotonos, is common. Paroxysms of delirium occur during which the animal stands with its head against the wall, or shows disorderly movements due to meningeal irritation. Coma and paralysis are followed by death in from five to forty-eight hours. In very acute cases death occurs in convulsions.

It seems probable that several diseases characterised by similar clinical symptoms have been confused by observers. M'Callum and Buckley found areas of softening in the frontal region of each side, anterior to the motor region of the cortex. Softening was practically confined to the white matter immediately under the cortex; in the areas affected there was complete destruction of the brain substance, the anatomical elements being disintegrated and largely replaced by colloid-like material. Neighbouring parts were acutely inflamed. In a second outbreak no softening was noted, but the condition of the blood-vessels indicated an early stage in the same process.

M'Carthy and Ravenel have studied the disease for some years, and unsuccessfully attempted to isolate an organism. Besides the lesions in the upper gastro-intestinal tract, where the infection probably occurs, the only others discovered were confined to the central nervous system, and may be grouped as follows :—

(1) Lesions of the intervertebral and Gasserian ganglia. (2) Lesions in the cerebral and cerebellar cortex. (3) Lesions in the choroid plexuses of the lateral cerebral ventricles. (4) Lesions of the peripheral nerves supplying the larynx.

The following changes were found in the intervertebral ganglia in nine cases. The capsule, consisting of a single layer of endothelial cells which closely surrounds the ganglion cells, and the supporting structure of the ganglion, composed of a loose areola of connective tissue, were all affected. The ganglion cells were the seat of extensive chromatolysis. The degenerative changes varied from a simple diffuse chromatolysis—a fusing together and loss of outline of the fine chromatin points in the cell protoplasm—to complete destruction of the cell body and nucleus. Certain cells appeared normal except for the accumulation of large amounts of a yellow pigment staining black with osmic acid. In several cells the nucleus was found displaced towards the periphery of the cell; in two cases the cell protoplasm showed marked vacuolation. In four cases some ganglion cells were completely disintegrated.

Capsular and Pericapsular Changes.—In all the above nine cases a pericapsular small-cell accumulation was present. Some of the degenerating ganglion cells still contained a few nuclei. The accumulation of nuclei around the cell capsule did not always assume a concentric shape, but was often eccentric, extending irregularly into the stroma. The cells were all of the small type, the nuclei and protoplasm being about the size of a red blood corpuscle.

Cortical Lesions.—The cortex of the cerebrum and cerebellum was markedly congested; the meninges were normal. Numerous capillary hæmorrhages were scattered throughout the entire cortex of the cerebrum and cerebellum. There were also hæmorrhages in the subcortical tissues. Except for some congestion of grey matter, the spinal cord was normal, as were the basal ganglia, pons, and medulla.

Lesions of the Choroid Plexus.—In three of the cases the choroid plexus was changed from a filmy membrane to a triangular tumour-like mass, of yellowish-red colour, of firm consistency, and one inch thick on transverse section. Microscopic examination showed this to be due to proliferation of the elastic tissues surrounding the vessels. The entire section consisted of whorls of delicate fibres extending from near the vessel walls to the margin of the plexus. The fibres were not nucleated, although numerous nuclei of the supporting tissue of the gland were present between the whorls.

The Peripheral Nerves.—The nerves supplying the larynx and neck showed slight degeneration, and this was present in the nerve up to the ganglion, but not in the posterior roots or the root of the fifth nerve.

Summary.—Hæmorrhagic inflammation of the upper respiratory organs; degeneration of the peripheral nerves supplying these areas; toxic irritation of the intervertebral ganglion, manifested by intense degeneration of the ganglion cells, pericapsular round-cell infiltration and swelling of the axis cylinders; wide-spread capillary hæmorrhagic extravasation of the cortical and subcortical tissues; tumour formation due to proliferation of elastic tissue of the choroid plexus of the lateral ventricles.

Conclusions.—(1) The so-called epidemic cerebro-spinal meningitis of horses is not a true meningitis, and presents neither the gross nor the microscopic lesions of true meningitis.

(2) The evidence goes to show that all epidemics are caused by some poisonous substance contained in the forage.

(3) The lesions in the intervertebral ganglia closely resemble those in rabies, and it appears that the pathological process in the two diseases is somewhat similar.

(4) The differential diagnosis between forage poisoning and rabies depends upon (*a*) The absence from the medulla and pons in forage poisoning of the pericapsular and pericellular lesions (rabic tubercles of Babes); (*b*) In forage poisoning there is a predominance of pericapsular rather than intracapsular round-cell infiltration of the ganglion cells; (*c*) Lesions of the larynx and laryngeal nerves. The clinical history is always conclusive.

(5) "Forage poisoning" is a much better and more comprehensive term than cerebro-spinal meningitis, or than leuco-encephalitis, as proposed by M'Callum and Buckley (The *Journal of Medical Research*, No. 77, October 1903, p. 243).

PUNCTURE OF THE BLADDER THROUGH THE RECTUM.

In over-distension of the bladder the two recognised methods of affording relief. are, firstly, ischial urethrotomy, and, secondly, passage of the catheter. The first is rapid, but often results in infiltration of neighbouring parts by urine and fistula formation, and is only effective when the obstacle to the passage of urine is below the point of incision.

Passage of the catheter, on the other hand, is in the horse sometimes difficult, and in the ox impossible. Matrion proposes puncturing the bladder through the rectum, which he declares to be easily carried out, efficient, and unattended by permanent bad effects. He uses a capillary trochar twelve inches long, and rather thinner than that employed for puncture of the cæcum. Such a trochar can be employed both for the horse and ox, and, being slightly curved, it is easily introduced into the bladder. It is used as follows: The rectum is evacuated and washed out with an antiseptic, the hand carrying the trochar is then introduced, the point covered with the index finger, and the instrument pressed into the most prominent portion of

the distended bladder. The point selected is on the median line and about the centre of the viscus. The vesiculæ seminales, vas deferens, and prostate are all thus avoided.

The instrument is introduced without violence, and is pushed forward until resistance ceases. The tissues are sometimes thickened and œdematus, and the space to be traversed is often considerable. Urine should be allowed to escape slowly in order to avoid possible reflexes which might cause respiratory or cardiac syncope. The operation may be repeated several times per day. If the obstacle in the urethra or bladder resists other treatment, time can thus be gained in which to attempt urethrotomy, cystotomy, or lithotrity. In the horse the posterior half of the bladder is not covered by peritoneum, but there is nothing to fear even in puncturing the anterior half, although the instrument then passes through a peritoneal cul-de-sac.

The operation has been performed on thirty-three subjects; twenty-seven horses, three oxen, and three rams. It is valuable in spasm of the neck of the bladder. in cases of urethral or vesical calculus, stricture of the urethra, œdema of the sheath, phymosis, or paraphymosis. In cases of calculus in oxen, where extraction appears dangerous or impossible, operation allows the animal to be kept sufficiently long to be sold for slaughter (*Revue Générale de Médecine Vétérinaire*, March 1903, p. 325).

PRIMARY TUBERCULOSIS OF THE UDDER IN THE COW.

ALTHOUGH tuberculosis of the udder is almost always secondary, some cases of primary infection have been reported. Conte describes the following one.

The cow appeared perfectly healthy and in good condition, the skin was loose, the appetite undiminished, and neither temperature nor respiration was increased. The superficial lymphatic glands were normal. Percussion and auscultation of the chest revealed nothing unusual. The two anterior quarters and the right posterior quarter of the udder were supple and elastic. The left posterior quarter was increased in size, and showed diffuse insensitive swelling, of fibrous consistence, affecting the whole of the glandular tissue. The skin covering this swelling was slightly tense, and concealed some small, hard nodules sunk in the superficial portions of the udder. The three healthy quarters of the udder yielded an abundance of normal milk. The affected quarter gave a yellowish serous fluid, holding in suspension greyish grumous masses containing tubercle bacilli. Injection of tuberculin caused a rise in temperature of 1·7° C., and marked diminution in the secretion of milk.

The history showed the disease to have existed for at least three months.

Except for a slight invasion of the parietal pleura, the lesions found on *post-mortem* examination were entirely confined to the udder. The diseased quarter weighed 7 kilogrammes, 550 grammes, contained very large numbers of caseous and confluent tubercles, and had become almost entirely converted into a fibrous mass. The mucous membrane lining the galactophorous sinuses was healthy, while that of the finer milk canals was covered with small yellow caseated granulations; the supra-mammary lymphatic gland was enlarged, and contained tuberculous lesions towards its centre. The extensive disease of the mammary gland and the trifling lesions in other portions of the body seemed to indicate primary infection of the gland itself. The owner had for some time been using as litter straw taken from palliasses removed from certain hospitals; the straw had probably been soiled by discharges from tuberculous patients.

Nocard has shown that, when functionally active, the udder is susceptible

to microbic infection, and that injection into it of tubercle bacilli without any mechanical injury of the gland always produces tuberculosis. Conte thinks that contaminated fragments of straw may have entered the teat, and thus caused infection of the gland (*Revue Générale de Médecine Vétérinaire*, March 1903, p. 327).

A BACILLUS RESEMBLING THAT OF TUBERCULOSIS FOUND IN BUTTER.

IN March 1899 Binot inoculated guinea-pigs with twenty-nine samples of butter obtained from different sources. Various results were obtained, but only in one case did an inoculated guinea-pig die spontaneously. This animal became very thin and perished in twenty-nine days.

On *post-mortem* examination its viscera were found normal in size and external appearance, except the spleen, which was slightly larger than usual. The liver and spleen, however, were crammed with fine miliary granulations, the size of a small pin's head, yellowish-white in tint, and vaguely suggesting the lesions produced by the cocco-bacillus. The peritoneum contained numerous miliary granulations, and some large masses with caseous contents and of considerably greater volume than the butter injected. The lungs and kidneys externally appeared normal, but exhibited a few miliary granulations.

The miliary granulations and the caseous masses referred to contained numerous bacilli presenting all the characteristics of the tubercle bacillus, and staining in a precisely similar way.

Attempts to obtain cultures from the blood were unsuccessful. With considerable difficulty cultures were finally obtained on glycerinated media. Isolated colonies on glycerinated agar at first appeared white, but soon became straw-coloured, then orange, and finally a deeper tint, resembling that of dried orange peel. In some instances the colony was as large as a florin, appeared brilliant, opaque, viscous and glairy on the surface, and was very adherent to the culture medium, without, however, extending into its depth. The surface was sometimes wrinkled and the margins raised. The colour was best seen in cultures exposed to light and oxygen.

On glycerinated potato the culture was abundant, homogeneous and opaque, at first straw-coloured, and then orange. Growth was less abundant on simple agar and on ordinary potato media. In ordinary bouillon no growth took place at first; later the microbe developed as a thin, friable, light yellow film, the liquid remaining clear, and a deposit collecting at the bottom of the vessel. Grown in glycerinated bouillon, the liquid remained clear, but its surface became covered with a thick, creamy, very viscous, homogeneous film. This film grew upwards on the edges of the flask. From its lower surface depended long glairy growths which floated in the liquid. The culture, although much more viscous and moister than that of avian tuberculosis, nevertheless resembled it, but with this difference, that at the end of some weeks it assumed a deep orange colour.

Under the microscope the growth was seen to consist of fine bacilli with rounded ends and of very varying length, the majority measuring 3-4 μ. The bacillus stained precisely like the true tubercle bacillus, and was "acid-fast," *i.e.*, resisted decolorisation by acids.

Two guinea-pigs were inoculated into the peritoneal cavity with the first bouillon culture. One survived; the other, which died in fifteen days, exhibited some miliary tubercles containing the microbe in the spleen and liver. A white mouse inoculated in the peritoneum died with a few discrete tubercles in the spleen (*Archives de Parasitologie*, May 1903, p. 306).

CUTANEOUS LYMPHANGITIS, AMYLOID LIVER, AND LARGE WHITE KIDNEY IN THE HORSE.

RIES describes the following interesting case. The subject was an eight-year-old cart-horse gelding, doing heavy draught work, and which in the ordinary course was obliged frequently to pass through a small stream.

When four years old this horse had shown lameness of the left hind leg, which was advanced very slowly, the animal making a cringing movement of the affected quarter at every step. The seat of lameness was not discovered. The animal was rested and the lameness disappeared.

Four years later, in the month of May, it began to exhibit the signs of the disease from which it eventually died. At first the sheath became swollen, and the animal showed lamenese of the left hind leg, particularly on leaving the stable. The appetite became capricious and was gradually lost. The horse fell away in condition and lost spirit. Ries saw the animal eight days after the first symptoms.

Seven or eight little abscesses had developed in the skin of the sheath, some were ready to break, others had already discharged, leaving small ulcerations in process of healing. Similar abscesses could be found here and there on the inner surface of the hind limbs, from the hoof as high up as the perineal region, scattered regularly along the course of the lymphatics, without, however, forming cords or true chains. One of these abscesses, in the flexure of the hock, had become tran-formed into an ulcer resembling that of farcy. All the abscesses, however, breaking, discharged "laudable" pus, and the resulting ulcers rapidly cicatrised. Irregularly distributed over the whole surface of the body, but few in number, were lymphatic cords, intradermic swellings, and little abscesses about to open, or already open and commencing to heal.

The submaxillary lymphatic glands were adherent to the jaw, enlarged (to a greater extent on the right than on the left side), slightly doughy, insensitive, and without peripheral infiltration. They did not suppurate. The nasal mucous membrane was red, a condition which appeared peculiar considering the general weakness.

The ilio-spinal muscles and those of the quarters were greatly wasted. The animal showed no pain when a man who was riding it allowed his entire weight to fall on the region of the loins. Nevertheless, it showed great weakness of the hind quarters. The hind limbs were dragged and adducted at the moment of leaving the ground. There was marked difficulty in advancing one of the hind limbs, and the corresponding quarter was depressed at each step. The animal knuckled over frequently, and its hind quarters rolled from side to side.

The rectal temperature was 38·3° C., the respiration slow and regular, circulation normal; the heart sounds were rather clearer than usual, and slightly metallic; the pulse was large and strong.

Pus from the enlarged lymphatic vessels was of a pinkish tint, from the abscesses a creamy yellow.

The urine was yellowish, transparent, and slightly acid; it contained neither albumen, sugar, nor bilirubin, and its only abnormal constituent was a few flattened epithelial cells.

The animal was placed on milk diet and given five drachms of salicylate of soda per day. The general condition improved slightly, but in eight to ten days this treatment was stopped and the disease made rapid progress. The wasting and weakness became more marked, and a discharge, at first from the right and then from both nostrils, set in.

Two months after the first symptoms appeared iodide of potassium was

given ; after five days of this treatment the urine became red, and was found to contain red and white blood corpuscles together with flattened epithelial cells. On rectal exploration the posterior margin of the left kidney was felt to be thickened and enlarged, so that it extended towards the pelvis and was clearly of considerable size. The right kidney did not seem affected.

Two days afterwards the animal seemed unable to move its jaws ; food accumulated between the molars and cheeks, though liquid could still be swallowed. The breathing sounds were very loud, the nostrils were dilated, and the animal appeared "tucked up" in the flank. Nasal discharge was abundant. Rectal temperature 38·7° C. A fatal issue being imminent, the animal was bled to death.

On *post-mortem* examination the following appearances were noted :—

The skin contained numerous small abscesses, the majority about the size of a hazel nut. The mucous membrane of the nasal cavities, facial sinuses, and larynx was covered with a whitish creamy exudate. Except for the œdema, particularly marked in the neighbourhood of the vocal cords, this part of the respiratory mucous membrane showed no change. The bifurcation of the trachea was covered with an exudate of irregular form, extending into the left bronchus for a distance of over 2 inches ; this exudate was membranous in character, greenish-yellow in colour, dry on the surface, and with sharply-defined margins. On removing it the mucous membrane was seen to be granular and suppurating. A more deeply excavated ulcer, the size of the end of a man's thumb, was found at the level of the last ring of the trachea. The lung exhibited acute diffuse vesicular emphysema, with trifling hypostatic congestion on the left side. The lobes were soft, elastic, and pale, and did not collapse either when the thorax was opened or they were incised. Under the pleura covering the median surface of the right lung was an abscess as large as a pigeon's egg, full of creamy white pus. The muscular wall of the left ventricle was hard and greyish, and was undergoing sclerotic change.

The most marked changes were found in the liver, which exhibited lesions of perihepatitis ; the portions of peritoneum opposite the gland were inflamed, the margins were rounded, and the whole organ was enormously enlarged, weighing 29 kilogrammes. On incision, the parenchyma appeared greenish-yellow, and showed the marbling peculiar to nutmeg liver, this marbling being of a deep reddish-brown tint. In consistence it was soft, pasty, but not waxy.

The right kidney adhered very strongly to the liver, in consequence of the perihepatitis already noted. Both kidneys were equally hypertrophied, the left being 27 cm., the right 23 cm., in length. They appeared pale, as though macerated in water. On section the parenchyma seemed lardaceous ; the cortical substance was grey, the medullary red. The pelvis of the kidney contained a brownish sediment with a violet metallic sheen. The medullary substance was infiltrated with the same colouring material (hæmoglobin). Microscopic examination revealed the absence of any fibrous change. Some uriniferous tubules were filled with a homogeneous exudate which had not taken the colouring matter. The epithelium of the convoluted tubules had undergone granular degeneration, the superficial cells being swollen, their protoplasm granular, and some had burst ; the deeper seated cells showed bosselated margins and were irregular in shape and less sharply defined than those of the normal parenchyma. The glomeruli did not otherwise appear changed. Altogether the lesions were those of enlarged white kidney.

On microscopical examination sections of the liver revealed granular degeneration of the hepatic cells ; there was no cirrhosis.

Tested with solution of iodine in iodide of potassium, followed by application of sulphuric acid, the tissues gave the amyloid reaction (*Receuil de Médecine Vétérinaire*, 15th October 1903, p. 629).

NOTE ON THE THERAPEUTIC USE OF
METHYLENE–BLUE.

BIDAULT commenced using methylene-blue in 1898 for flap wounds with extensive separation of skin, which prove difficult to heal by ordinary antiseptic methods.

Although the substance is somewhat expensive, costing about 50 francs per kilogramme, its use in such cases is quite practicable, inasmuch as the solutions employed are not concentrated. A saturated solution in water contains 4·57 per cent. The living cells have the power of reducing it and thereby destroying its staining quality.

Methylene-blue is antiseptic. In saturated solution it rapidly destroys staphylococci, streptococci, the bacillus coli communis, and the anthrax bacillus. On the other hand, some microbes, like Eberth's bacillus and Friedländer's bacillus, resist even prolonged contact. Bidault declares that a 1 per cent. solution destroys staphylococci in twenty-four hours. Methylene-blue is not caustic, and in human medicine is said to have analgesic properties which render it suitable for diminishing pain in neuralgia, enteritis, and rheumatism. Injected like cocaine, it produces in about ten minutes a sufficiently marked analgesic effect to relieve painful lameness. Such injections, however, are followed later on by painful enlargement, which persists from four to six days.

Bidault suggests that methylene-blue might be used in the treatment of acute rheumatic lameness, and enteritis with obstinate diarrhœa. He treated a young horse suffering from rheumatic synovitis of the great sesamoid sheath which had resisted all ordinary methods. Doses of 50 cm. of powdered methylene-blue were given in gelatine capsules. The dose was repeated three times a day. Treatment was commenced on the 1st May and by the 5th of that month lameness had almost disappeared. The dose was then increased to 2 grammes per day until the 8th May, after which 1 gramme per day was given for the next week. On the 18th lameness had disappeared. Since that time the horse has done military service without showing any signs of lameness.

Bidault claims to have had good results from the external use of 1 per cent. solution in cases of conjunctivitis.

For wounds he recommends 2 per cent. solutions. The parts are thoroughly cleansed and washed with boiled water, and then cautiously injected or mopped with a solution of the above strength. He also claims to have had remarkably good results in the treatment of synovitis and open arthritis with 2 per cent. solutions. A troop horse in which the great sesamoid sheath had been deeply penetrated was cured in ten days by injections. In another case, affecting a thoroughbred horse, the synovial sac of the tibio-tarsal joint was accidentally perforated when racing. Synovia escaped in considerable quantity, and in spite of the careful use of sublimate solutions the synovial capsule became extremely tense and the general symptoms more and more marked, until, after two days of treatment, recourse was had to injections of methylene-blue. Five days later the discharge ceased. Harness injuries are treated with 2 per cent. solutions of methylene-blue in glycerine. Bidault speaks highly of the results obtained (*Rec. de Méd. Vétérin.*, 15th November 1903, p. 697).

TRAUMATIC PERICARDITIS IN SWINE.

CASES of traumatic pericarditis in swine are extremely seldom seen, and interest therefore attaches to a case of the kind described by Teetz. He was called to inspect a pig which, it was stated, had been in indifferent health for

about six weeks. Sometimes it ate well, and then for a time ate scarcely any-
thing. Nevertheless, it did not markedly lose condition, for at the time of
examination it weighed about 180 lbs., its companion in the stye weighing
about 210 lbs.

The condition on examination was as follows: The pig lay almost
insensible, and could only be made to rise with difficulty. The skin covering
the ears, the parts behind the ears, the larynx, breast, abdomen, and the inner
surfaces of the hind limbs were of a bluish tint. Teetz suggested slaughtering
at the earliest possible moment, and suspected chronic swine plague or tuber-
culosis.

The *post-mortem* revealed the following: The lungs and some of the
mesenteric glands were tuberculous. No other tuberculous changes were
found in any of the organs or of the lymphatic glands. The slaughterman had
incised the pericardium when cleaning the animal, and had allowed a large
quantity of an ill-smelling fluid to escape. The walls of the pericardium were
greatly thickened, the outer layer being ¾ cm. in thickness; the colour of the
parts was greyish-yellow and therefore very striking. On the epicardium were
numerous warty growths and deposits which could be stripped off in layers with
the knife, and then appeared as thin fatty membranes consisting of a crumb-
ling, greyish-blue ill-smelling material. The entire epicardium was covered
with a greyish-yellow coating which could be stripped off the heart with the
fingers. At the base of the heart the visceral and parietal layers of endothelium
were adherent. The heart was now divided, exposing the cavities. In the
outer wall of the right heart was a blackened, rough sewing-needle about 2
inches long, and lying in a nearly horizontal position (supposing the animal to
be standing). A yellowish black tract could be detected below the endocar-
dium, which on being cut into proved to be the channel produced by the
passage of the needle. It was surrounded by a yellowish capsule about 2
mm. thick. Capsule formation was incomplete at the point last reached by the
needle, and the endocardium presented instead a tubular raw wound of a green
tint. The needle was crumbling, and on being grasped with the fingers broke
in two.

It is noteworthy that the pig had shown no trace of subcutaneous œdema,
such as is usual in oxen under similar circumstances. The blue coloration of
the skin at the points above mentioned had only developed during the few
days preceding slaughter, and was no doubt due to defective heart action
(*Zeitschrift f. Fleisch und Milchhygiene*, November 1903, p. 61).

PUNCTURE OF THE COLON PER RECTUM.

ELSCHNER has practised puncture of the colon per rectum for the past eight
years. On *post-mortem* examination of a horse which recently died he had
great difficulty in finding any indication of the wound in the rectum. The
zone of inflammation surrounding it was scarcely noticeable. This induced
him to make further experiments, and, although regarding puncture per rectum
as a last resort, he has since performed the operation on horses a number of
times without bad results. He mentions three cases. In the first, which
suffered from obstinate tympanites, he used the trocar three times; once on
the left side, once on the right, and lastly through the rectum. The horse
recovered, though only after ten days. In the second case he operated first
from the right flank and then from the rectum. The horse recovered. In
the third case he operated twice through the rectum. The horse recovered,
though it continued to show trifling fever with small frequent pulse and
increased respiration for a week. Elschner, however, refers these symptoms
to absorption of toxic products from the bowel and not to the operation.

There was no swelling around the site of operation in the rectum, or pain on defæcation or on manual examination.

He therefore regards this operation as of a great value, although not entirely free from danger. Nevertheless he has successfully performed it several times without disinfecting the rectum or trocar, the rectum being simply washed out with clean water and the trocar placed in carbolic solution. Whenever possible he recommends washing out the bowel with ½ per cent. lysol or creolin solution and immersing the trocar and canula in a 3 per cent. solution of the same character. Formerly he used the trocar intended for tapping the chest, but as he found it too short for convenient handling he has for some years employed a special trocar with a 16 inch canula. The shield is fixed about 1¼ inches distant from the handle, so that if necessary a rubber tube can be affixed for the purpose of injecting fluid. The point of the stilette extends nearly an inch beyond the canula, which is 5 mm. in diameter. He would prefer a narrower trocar were it not that this would render the instrument too pliable. On account of the double thickness of intestine to be pierced the stilette must extend about 1¼ inches beyond the canula. The entire instrument might with advantage be made 18 inches in length (*Berliner Theirärztliche Wochenschrift*, 7th January 1904, p. 27).

MALIGNANT CATARRHAL FEVER OF THE OX.

IN almost all treatises dealing with this disease the symptoms are described as follows: At first rigors, staring coat, loss of appetite, constipation, high temperature, cessation in secretion of milk, dulness intermitting with periods of excitement. irregular distribution of surface temperature, abnormal warmth around the base of the horns and around the coronets, stiffness in moving, marked redness of the visible mucous membranes of the head, and slight discharge from the eyes, nose, and mouth ; on the second or third day severe swelling of the eyelids and marked lachrymation, photophobia, turbidity of the cornea, severe salivation. muco-purulent discharge from the nose, and marked falling off in the general condition ; two or three days later slight increase in the turbidity of the cornea, croupous-diphtheritic deposits on the respiratory mucous membranes, rattling breathing, diarrhœa, difficulty in urination, croupous diphtheritic deposits on the vagina, eruptions on the surface of the skin, loss of strength and body weight, continued lying down, loosening and ultimate loss of the horns, convulsions, and death. Another form of the disease, which lasts longer and is accompanied by frequent relapses, has been described. The prognosis is regarded as unfavourable.

The *post-mortem* appearances are in accordance with the symptoms : keratitis, extravasations into the chambers of the eye, turbidity of the lens, croupous-diphtheritic deposits on, and ulceration of, the mucous membranes of the respiratory, digestive, genital, and urinary organs, congestion of the brain and its envelopes, hæmorrhages. enlargement of the mesenteric lymphatic glands, and thickish but fluid condition of the blood.

The diseases with which malignant catarrhal fever may be confused are as follows: Rinderpest, tuberculous basilar meningitis, dysentery, periodic ophthalmia, and rhinitis fibrinosa.

As in all diseases which usually prove fatal, many different methods of treatment have been recommended, comprising bleeding, the use of hellebore in the form of plugs inserted under the chin, medicated inhalations, the administration of tartar emetic, nitrate of potash, etc., sawing off of the horns, washing with vinegar, application of poultices to the head, trephining, the administration of creolin, carbolic acid, and antipyretics by the mouth, and the intravenous injection of argentum colloidale, etc.

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Regarding the cause of catarrhal fever, most authors agree in believing that the disease only attacks bovine animals, is infectious, though very slightly or not at all contagious; that it rarely assumes an enzoötic form but is always sporadic; that the cause is a micro organism, different, however, from that of diphtheria; that dirt, moisture, and want of drainage favour the disease; that young and well-nourished animals are most subject; that the disease is commonest in spring and occurs at all elevations; finally, that the cause previously suggested, viz., the action of cold, has only a predisposing effect.

The period of incubation is from three to four weeks.

The exact cause of the disease is still unknown.

In the Canton of Graubünden 45,000 animals are usually insured. In the year 1898, 39 of these died of the disease; in 1899, 57; in 1900, 109; in 1901, 96; and in 1902, 106; altogether 407 beasts. In Zürich about 94,738 animals were insured, and of these only 5 were attacked by the disease. In 1900 some 90,855 were insured, and of these only 3 were affected.

The writer describes several cases both of a typical and an atypical character, particulars of which were supplied by his colleague, Meissen of Disentis. It is remarkable that amongst all these cases only one displayed the typical symptoms of malignant catarrhal fever, the others exhibiting a mixture of symptoms which rendered the diagnosis of the disease extremely difficult. This one animal showed the following appearances : sudden onset of diarrhœa, the dejections being thin, of a repulsive odour, and often blood stained; high fever, the temperature rising to 42° centigrade; quiet breathing. slightly accelerated pulse, loss of appetite, great thirst, slight reddening of the visible mucous membrane, dulness alternating with periods of restlessness, staring gaze, watery discharge from the eyes, transparency of the cornea, change in the shape of the bulbus, the cornea being thrust forward in a pointed form indicating pressure in the postocular space. After the second or third day the animal seemed in constant danger of sudden death, especially when forced to move.

On *post-mortem* examination the mucous membranes were found to be only slightly redder than normal, the lungs were œdematous, the blood was dark, the heart dilated, the brain œdematous and dotted with apoplectic points, the stomach and bowels exhibited signs of intense gastro enteritis, the liver was friable and the gall bladder enlarged, but the spleen appeared almost normal. This might be regarded as a peracute form of catarrhal fever, similar to the peracute forms of foot-and-mouth disease and of diphtheria in man, where the micro-organisms appear so virulent and the formation of toxins so rapid that death occurs before characteristic signs of the disease have time to develop.

Regarding the treatment of this disease, we learn from textbooks that 50 to 90 per cent. of the animals attacked died. The writer has often attempted treatment, in some cases employing intravenous injections of argentum colloidale, but has only saved one animal which showed severe symptoms. Even then the patient proved worthless from the economic standpoint, for although it certainly lived it was blind, had lost its horns and hoofs, was in poor condition and gave no milk : in a word, its value was less than the cost of treatment. In the early stage, however, it seems possible, by free bleeding, to check the course of the disease, provided no marked changes have taken place and the symptoms are confined to roughness of the coat, severe fever, peculiar staring appearance of the eyes, and slightly increased prominence of the cornea. The writer is well aware that in infectious diseases bleeding has been condemned by many authors of experience, but states, as a result of his observation, that in malignant catarrhal fever it often proves of great benefit.

He recommends thorough disinfection of all places in which infected

animals have been kept and careful attention to drainage. His conclusions may be summarised as follows :—

(1) Malignant catarrhal fever is a disease almost entirely confined to bovines, is not transmissible from animal to animal, but is produced by an organism present in the soil, probably a form of bacterium coli. It possesses great powers of resistance outside the animal body.

(2) Malignant catarrhal fever often assumes so extremely acute a course that it can only be confidently diagnosed in places where typical outbreaks have occurred.

(3) It usually assumes a sporadic form, but may also occur enzoötically. It attacks animals of all ages. Sheep and goats are not immune against it.

(4) It occurs at all periods of the year, but most commonly in spring, and is more frequent in hilly regions than in plains, though its occurrence seems to have no connection with the geological formation of the sub-soil.

(5) The disease may be confused with a number of others. In its initial stages it closely resembles foot-and-mouth disease, and in its peracute form rinderpest, meningitis, and dysentery.

(6) The flesh of animals slaughtered before the appearance of the croupous-diphtheritic deposits is not dangerous to health, and in some cases may be exposed for sale in the ordinary way.

(7) Once well developed, the disease is fatal. In practice one usually proceeds as follows : Provided no typical symptoms have developed, free bleeding will often lead to improvement and recovery, but should no improvement occur within the following twenty-four hours it is advisable to slaughter the animal in order to prevent injury of the flesh.

(8) To prevent the disease spreading all diseased animals should be removed from the infected byre, which should be disinfected as after anthrax. Lorenz's method of serum treatment, on the principles laid down for treating swine erysipelas, would probably prove the most successful system of treating this disease, but the relative rarity of malignant catarrhal fever and the difficulty of preparing and storing the serum are so great as to remove this suggestion almost beyond the region of practice (Isepponi. *Schweizer-Archiv für Thierheilkunde, XLVI. Band., 1 Heft.,* 1904, p. 1).

FOWL PLAGUE.

Until recently all enzoötic or epizoötic diseases of birds characterised by rapidly fatal development, somnolence, diarrhœa, and dark coloration of the mucous membranes and crest, were generally regarded as fowl cholera. The differentiation of the numerous septicæmias of birds scarcely modifies the position as regards diagnosis and prophylaxis. All are due to ovoid bacteria related to the form typical of cholera, and the choleraic infections which they produce seldom show more than trifling peculiarities among themselves; all call for similar prophylactic measures.

In 1880, however, Rivolta and Delplato studied a disease of poultry which they distinguished clinically from cholera and named exudative typhus, a term which both suggested the gravity of the general condition and the presence of inflammatory exudates in the body cavities, particularly in the peritoneal cavity.

Between 1894 and 1900 this atypical choleraic disease was seen in almost all parts of Central and Northern Italy, where it assumed the form of an epizoötic. Some observers regarded it as a special type of fowl cholera, others isolated various bacteria which they believed to be specific.

The solution of the questions thus raised was afforded by Professor Centanni

of Ferrara and his pupil Savonuzzi.   In March 1901 they showed that the·
bacterium of cholera is never found in the new disease, and that the virulent
liquids contained no microbe detectable by existing bacteriological methods.

They named the disease fowl plague (peste aviaire), a name which it has
since retained.   In the following month the same authors discovered certain·
properties of the virus, particularly its power of passing through porcelain
filters and the impossibility of cultivating it on artificial media.

From this time it was clear that the disease, like contagious pleuro-
pneumonia, cattle plague, foot-and-mouth disease, and horse sickness, was·
due to an ultramicroscopic agent.

The disease specially attacks fowls, other birds in the same yard often
remaining unaffected.   Pigeons, which are so sensitive to fowl cholera, success-
fully resist contagion.

The course of the outbreaks is similar to that of fowl cholera.   As a rule
the poultry yards are swept clean in a week ; sometimes, however, the disease
continues for several weeks.

The affected bird seems depressed, refuses to eat, retires from its fellows,
and remains "bunched up" and still.   These symptoms become more marked
hour by hour.   The bird is sleepy, its eyes are half closed, the feathers
ruffled, and it makes frequent swallowing movements.   Expiration is full,
difficult, and accompanied by rattling sounds ; the temperature rises to
43° or 44° C.

A little later coma sets in ; the bird looks like a ball of feathers, the chest
and abdomen rest on the ground, the feet are spread out on either side ; the
bird staggers and falls if disturbed.   It cannot be aroused, whatever means be
adopted ; vision seems lost, mucus escapes from the beak and nostrils, respira-
tion becomes dyspnœic, the crest and mucous membrane assume, first, a
reddish-brown, and afterwards a violet, tint.   As a rule the excrement is of
normal colour and consistence, though some birds pass greenish liquid.

The last period is marked by progressive paralysis, commencing in the
posterior parts of the body or in the limbs.   Some cases show circus or roll-
ing movements, unilateral paralysis of the limbs or neck, or, again, up and
down movements of the head and neck.

Death almost always occurs one to three hours after onset.   In exceptional
cases the birds may survive five to eight hours.

The lesions are similar to those of infectious toxæmias.   The blood is
liquid ; the parenchyma of the internal organs is softened ; the liver is yellow,
friable, and has undergone fatty degeneration, and the mucous membranes show
ecchymoses.

None of the local changes mentioned by different authors are constant.
Hæmorrhage is particularly frequent in the mucous membranes of the stomach,
where it occurs between the masses of gland tissue, rare in that of the small
intestine, cloaca, mouth, and eyelids.   The distribution of the ecchymoses and
exudates in the serous cavities is also irregular.   The peritoneal, pleural, and
pericardial cavities may contain sero-fibrinous exudates or a turbid liquid
resembling milk.

Some birds may show no changes on superficial examination.

The epidemic character of the disease, the rapidity of development, and the
"typhoid" conditions limit the diagnosis to fowl plague or fowl cholera.

The distinctive peculiarities consist in the constant occurrence of diarrhœa
and inflammatory lesions of the intestinal mucous membrane in cholera,
though in practice these signs have only a very relative value.   Of greater
diagnostic value is the fact that plague is limited to fowls, and that pigeons
resist it absolutely although they are common victims of fowl cholera.

The diagnosis is confirmed by the discovery of the bacteria of cholera in the·
blood or in the parenchyma of the internal organs, or more simply still by

inoculating a pigeon ; in the case of cholera this pigeon would die in twelve to thirty-six hours, in that of plague, however, it remains unaffected.

The virus exists in all parts of the dead body. It can be found in the secretions of the mucous membrane and in the intestinal dejections.

The virulent liquids retain their properties after filtration through a Pasteur-Chamberlain or Berkefeld filter. The activity of the virus is considerable. Solutions of one part diluted with 125 million parts of water kill fowls in forty-eight hours when injected in doses of 48 cc. The organism has not been cultivated on any artificial medium, and cannot be obtained *in vivo* by Nocard's method, *i.e.*, sowing in bouillon contained in a collodion sac which is afterwards inserted in the peritoneal cavity of a living fowl.

Inoculation or ingestion of the virus is very fatal to fowls and also kills the turkey, guinea-fowl, goose, and duck, though with less certainty. Pigeons and rabbits are almost proof, and can only be killed by large doses. Other animals are refractory.

Contagion is undoubtedly effected through the medium of the virulent dejections. It seems less easily diffused than that of fowl cholera. The virus is very delicate and, unlike the ovoid bacteria, does not live long in the soil and water, though any such drawbacks are compensated for by its extreme powers of infection.

The virus is relatively insensitive to dessication. Dry blood is still active after twenty to twenty-five days. On the other hand, the virus is destroyed in a few moments in a temperature of 65 to 70° centigrade ; ·1 per cent. sublimate, 5 per cent. carbolic acid, creolin, lysol, etc., destroy the virus by simple contact.

As a rule, it is impossible to prevent the spread of infection once a fowl run has been invaded. Treatment is quite useless, and it is better at once to kill all the fowls. In some enzoötics where the disease spreads slowly, attempts may be made to save very valuable birds by isolating each one in a spot removed from the possibility of contagion. Disinfection is easily carried out by washing the sheds with potash soap, followed by watering with antiseptic solutions or using a steam jet.

The presence of fowl plague in a neighbouring fowl run necessitates precautionary measures. The fowls should be kept shut up, as far as possible, and care taken to prevent small birds, like sparrows, obtaining access to the run, as they form dangerous vehicles of contagion.

All newly purchased fowls should be kept in quarantine for a week. The importance of the losses caused by fowl plague would justify the enforcement of special sanitary measures if such measures could be shown to be practicable. The majority of the States forming the German Confederation have extended the law of 1880, dealing with contagious diseases, to fowl cholera and fowl plague.

When the disease is detected the owner is required to inform the police and to make a detailed report of all the birds he possesses. The bodies are to be burnt or boiled, or buried between two layers of quicklime. The police at once give notice to the sanitary veterinary inspector who confirms the diagnosis, gives directions as to the measures to be taken, and indicates the length of time during which the premises are to be kept under observation.

Notice is officially given of the existence of the disease in the administrative publications and posters which are displayed near infected establishments.

Neither dead nor live birds may be removed from infected localities.

Should a fowl when being sent by rail die under suspicious circumstances, the whole of the birds comprised in that consignment are seized and placed under observation, while the waggons are disinfected. If fowl cholera or fowl plague is detected the consignment is taken in hand by the sanitary officials.

Infected places are cleaned with hot 3 per cent. solution of soda. After

drying and exposure to the air they are whitewashed with freshly made 5 per cent. lime solution, and the troughs are filled with 1 per cent. chloride of lime solution.

Special directions have been laid down for the management of poultry shows (Léclainche. *Revue Générale de Médecine Vétérinaire*, No. 26, January 1904, p. 49).

THE EFFECT OF INJECTIONS OF COCAINE ON SEN-SITIVE NERVES, WITH SPECIAL REFERENCE TO THE DIAGNOSIS OF LAMENESS IN THE HORSE.

MM. TRASBOT, Butel, and Barrier having cast some doubt on the absolute reliability of the indications furnished by injecting cocaine along the course of sensitive nerves with the object of diagnosing lameness in the horse, M. Dassonville has taken up the subject and conducted a series of experiments. His investigations were based upon two principles, which he has enunciated in the form of laws. The first is as follows :—

When cocaine is injected along the course of the sensory nerves of a limb the sensation of pain in the peripheral regions served by these nerves is suppressed. Thus, if cocaine is injected along the course of the median and cubital nerves, pain should cease to be felt throughout the lower region of the radius of the corresponding side.

If the injection is made along the course of the plantar nerves, opposite the point of their anastomosis at the back of the metacarpus, sensation should be suppressed throughout the lower part of the region of the cannon bone, the whole of the fetlock, and the digital region.

If the injection is made at the point usually selected for high neurectomy, sensation should be lost in the pastern and subjacent parts.

If at the point where low neurectomy is performed, sensation should be lost in the foot.

In the different cases examined the seat of pain was identified by two different methods. In some the animal showed grave lesions which were evident to the senses (suppurative arthritis, picked-up nail, etc.) and were unquestionably the cause of the lameness. In others the seat of the lesions was obscured and was only revealed by injections of cocaine. In these instances the diagnosis was afterwards confirmed by performing neurectomy at different levels.

The experiments were therefore divided into two classes : (1) those in which the seat of lameness was known, and (2) those in which it was obscure.

In the fourteen experiments which were made, it was found that, whether the seat of lameness was known beforehand or was afterwards determined by the effects of neurectomy, the lameness disappeared whenever cocaine was injected above the lesion, whatever distance above the injury the injection might be made.

The total number of experiments undertaken in this way was twenty-five, and all gave identical results. M. Dassonville therefore considers that the law above enunciated is well established.

The second law which he lays down is as follows : When cocaine is injected along the course of sensitive nerves in a lame limb, but below the lesion which causes pain, the perception of that pain is in no wise impeded.

He supports this view by citing a series of cases. The first was that of an animal in which the perforatus tendon had been accidentally divided at the upper extremity of the first phalanx. After healing considerable enlargement remained, the animal proved lame, and was useless for work. Injections of cocaine were made along both plantar nerves at the points usually selected for

low neurectomy. Lameness persisted. A second attempt also gave negative results, but when injections were made at the points usually selected for high neurectomy the lameness entirely disappeared.

The second horse had a large splint, involving the lower row of bones of the knee as well as the large metacarpal and internal splint bone; lameness was intense. No improvement followed injections of cocaine along the plantar nerves at any point below the lesion, either at the seat of low or high neurectomy or at the point where the nerves anastomose at the back of the meta-carpus.

An injection along the median and cubital nerves caused the lameness completely to alter in character and to diminish considerably.

The third, fourth, fifth, and sixth cases were lame in the front legs, though the seat of the injury was unknown. Injections were made over the seat of low neurectomy; the lameness was slightly diminished but did not disappear.

Injections were then made over the seat of high neurectomy, over the post-metacarpal anastomosis, and along the course of the median and cubital nerves; in each case the lameness disappeared completely.

These results were interpreted as proving, firstly, that the seat of lameness was in the digital region, and that it extended beyond the area served by the nerve fibres divided in low neurectomy, but not beyond the area served by those divided in high neurectomy.

Secondly, that if it were desired to remove the lameness by neurectomy. the high and not the low operation was indicated. The operations were performed, the results completely bearing out the above conclusion.

The seventh and eighth animals were each lame in a front leg, and in both the coronet exhibited a large ringbone extending over the entire length of the pastern. To these ringbones the lameness was attributed.

Cocaine was injected along both plantar nerves at the site of low neurectomy, that is to say, at a point below that occupied by the great mass of the lesion; the lameness was in no respect modified.

Injections made at the site of high neurectomy caused the lameness completely to disappear.

High double neurectomy was then performed, and the animals became quite sound.

From these eight experiments M. Dassonville concludes that when cocaine was injected below painful points the area and position of which could be exactly determined by objective lesions like the wound on the pastern, the ringbones, splints, etc., or by the performance of neurectomy, the lameness was not modified. In every instance, however, when cocaine was injected above these lesions the lameness disappeared. From these and subsequent experiments he considers his second law to be well established.

He then proceeds to lay down a general method of detecting the seat of lameness in the horse. Given a lame horse, to determine the seat of lameness.

The primary injection should be made along the course of the median and cubital nerves.

- The lameness disappears; a second double injection should be made above the fetlock.
  - The lameness disappears; a third double injection should be made over the site of low neurectomy.
    - The lameness disappears; its seat is in the foot.
    - The lameness persists; its seat is in the first phalanx.
- The lameness persists; a third double injection should be made over the site of the plantar anastomosis.
  - The lameness disappears; its seat is in the lower part of the cannon.
  - The lameness persists; its seat is between the upper third of the cannon bone and the middle third of the fore arm—to be more exact, an injection should be made along the course of the cubital nerve; if the lameness disappears, the lesion is within the area served by this nerve.
  - The lameness persists; the lesion is in the scapular region, in the region of the arm or upper fore arm (practically the shoulder).

In applying the above method M. Dassonville has always found the indications furnished by cocaine in complete accord with those revealed by direct examination (in cases of objective lesions, like picked-up nail, arthritis), or by the performance of neurectomy (in cases where no visible lesions existed).

He has thus examined twenty-five horses, five of which were lame in both fore feet—in all thirty diseased limbs, and has made 108 double injections along the course of the nerves. These results therefore appear sufficient to eliminate the effects of chance and other causes of error.

In answer to M. Butel's objection that he (M. Butel) had been led to localise the lameness in the lower regions of the limbs because the lameness had diminished after a double injection along the plantar nerves, slightly above the fetlock, M. Dassonville points out that the injections should have been repeated at higher points until the lameness had entirely disappeared, and that it is quite possible for a horse to show lameness from two co-existing causes, such as simultaneous lesions in the foot and shoulder. As M. Butel did not repeat the injections at higher points, his objection to the reliability of cocaine carries no weight.

M. Barrier's objections were of two kinds, the first being that the uncertainty of diagnosis based on cocaine injections was well recognised by human practitioners, and secondly that the defects of the method were in no respect surprising to physiologists, bearing in mind the known facts regarding the phenomena of recurrent sensibility.

In answer to these objections, M. Dassonville pointed out that in man cocaine is not required for diagnostic purposes, it being always very much better to ask the patient where he feels pain, and therefore in man no one has

tested such injections with this object, that is to say, human practitioners have no basis for forming an opinion on this subject.

Reviewing the practice of prominent German, American, and French surgeons, M. Dassonville showed that no one was justified in declaring that injections of cocaine along the course of the nerves were condemned by medical men.

As regards the opinions of physiologists, Arloing and Tripier had shown that the peripheral stump of a divided nerve is sensitive on account of the peripheral recurrent fibres which follow the course of neighbouring nerves, and also that the cocaine acts on the direct fibres with which it comes in contact, but has no action on the recurrent fibres which, following the course of other nerves, may supply higher points in the neighbourhood.

It is true, that these clever experiments have clearly shown the existence of peripheral recurrent fibres.

On account of the existence of these fibres it is easy to understand why, in the horse, division of a plantar nerve rarely removes the perception of pain in the region apparently served by this nerve. In point of fact pain may be transmitted to the central nervous system by the intact plantar nerve through the intact recurrent fibres which originate in this region, but if one prevents such transmission by simultaneously dividing both plantar nerves the influence of recurrent fibres is completely destroyed and complete insensibility obtained. Thus the teachings of practice sufficiently demonstrate that after double neurectomy pain in the parts served by the two peripheral stumps disappears, provided one is careful to operate at a sufficiently high point to prevent there being any further direct relation between either central stump and the fibres supplying the injured part.

Now, the method above indicated produces results similar to those of a double neurectomy (necessarily of a temporary character), for each injection has a double effect; it not only interrupts the transmission of sensations from the region served by the direct fibres but also of sensations which are transmitted from the area served by the opposite nerve through the medium of the recurrent fibres.

The effect is, in short. to prevent any transmission of painful impulses. As a consequence M. Barrier's objections fall to the ground. They would be applicable were the injections made only on one side, but in point of fact they are always bilateral.

Furthermore, if the existence of recurrent fibres proved the unreliability of cocaine injections the same fact would also prove the unreliability of double neurectomy as a means of removing painful sensations, but even the most severe critics of neurectomy would scarcely dispute the anæsthetic effects of these operations.

Summing up, M. Dassonville declares that neither the general opinion of human practitioners nor the physiological objections put forward by M. Barrier in any way shake the principles which he deduces from the above described experiments.

Injections of cocaine will always enable one to detect whether the lesion producing a nervous lameness is localised in the foot, the pastern, the fetlock, the cannon, the knee, or the upper regions of the limb.

General Conclusion.—The value of the method of diagnosing the seat of lameness by injecting cocaine along the course of sensory nerves is proved partly by the 108 experiments carried out on 30 lame limbs, partly by the control experiments published by MM. Deysine, Vidron, Almy, and Pécus. It has not been damaged by any contradictory facts, and has not been shaken by the theoretical objections advanced against it. It remains entire.

In a subsequent discussion at a meeting of the Central Veterinary Society of Paris, M. Desoubry spoke as follows: "Before concluding I shall endeavour to show how strong the local anæsthetic action of cocaine is, and also to point out a rare but nevertheless possible danger of these injections."

"A five year old mare belonging to M. S——— of Versailes was sent to

grass on account of lameness in the off fore leg. The lesion, as shown by an injection of cocaine, was in the digital region.

"One Sunday afternoon I was requested to go immediately to see the patient, which had suddenly developed extremely severe lameness in this limb. The animal could bear no weight on the limb. The owner thought that in galloping it had strained itself. Nevertheless the sensitiveness of the coronary region. even in the absence of all crepitation, suggested to me a grave lesion, possibly a fracture of the second phalanx. To convince the owner I made a double injection of cocaine in the fetlock region. Shortly afterwards the mare walked and trotted with remarkable freedom. Three quarters of an hour later the lameness re-appeared with all its previous alarming symptoms. The diagnosis of fracture of the os coronæ was confirmed by M. Almy, and the animal was slaughtered. I produce the fractured portion of the bone for your inspection. The fracture was of a multiple character, which exactly corresponds with the symptoms observed.

"A horse belonging to M. P—— of Versailes, when returning from the farrier's, fell so lame that he walked on three legs. Examination of the limb and digital region did not at first reveal sufficient to justify a precise diagnosis. I then injected cocaine on both sides of the pastern. The lameness disappeared completely. Despite the various forms of treatment used, no improvement followed, and three weeks after the accident the owner consented to the animal being destroyed. On *post-mortem* examination the third phalanx was found clearly fractured and separable into two fragments on removal from the hoof. Here again cocaine had admirably served its purpose.

"In the *Revue Général de Médecine Vétérinaire* of the 1st March 1903, I find a case reported by Becker in Germany, which shows that injections of cocaine employed for diagnostic purposes may sometimes cause grave accidents. In this particular case a lame horse received an injection of cocaine over the plantar nerves: about an hour after lameness had completely disappeared the animal fractured the second phalanx of the limb in which the injection had been made. Eberlein cites an almost identical case. The horse fractured its first phalanx.

"In my own practice in August 1902 I injected cocaine in a case of obscure lameness. The injection gave positive results, and I localised the injury as in the digital region. The horse was sent back to the owner's stable, but in the afternoon a messenger came to inform me that it could no longer support weight on the injected limb. On my seeing it I found that the horse showed excessive sensitiveness over the entire region below the point of injection. There was no heat or engorgement. For about ten days the symptoms continued of this alarming character, but disappeared entirely after median neurectomy had been performed by M. Cadiot.

"In the cases reported by Becker and Eberlein one cannot give a satisfactory explanation of the appearances shown. The two horses in question must have suffered from disease of the bone which had caused changes in structure and prepared the way for fracture.

"Under ordinary conditions, animals proportion the weight which they place on the limb to the pain experienced, and thus protect the injured bone. When, however, an injection of cocaine has deprived the animal of sensation in the part it no longer appreciates the warning furnished by pain, throws more weight on the limb, and may thus fracture one of the phalanges which previously had been the seat of ostitis.

"In the case just described, the explanation is probably more difficult. The exaggerated sensibility was possibly due to a bone lesion which may have become aggravated when sensation was removed from the injected nerve. It may however have been due to an injury of the nerve itself inflicted at the moment of injection. I cannot say, but I consider that after an injection of cocain it is wise to keep the animals absolutely quiet until the anæsthetic action has entirely disappe red."

M. Desoubry spoke as follows: "The doses of cocaine which I recommend and which can be administered without any bad results vary between fifteen and forty centigrammes for ponies and large horses respectively. I have never seen toxic results. In introducing the needle a vessel may be pricked, but this is a trifling matter and need cause no anxiety. The needle should then be withdrawn and inserted at a spot where hæmorrhage is not to be feared. The injection never has serious consequences provided the most elementary precautions are observed. Such accidents as swellings or abscess formation are due to carelessness.

"Contrary to what has been stated by most writers who have dealt with cocaine, I obtain the anæsthetic effects in a relatively short time—five to six minutes. I think it useless to wait for fifteen to twenty minutes as some operators suggest" (*Receuil de Médecine Vétérinaire*, 29th February 1904 p. 80).

INTESTINAL COCCIDIOSIS OF YOUNG BOVINE ANIMALS (HÆMORRHAGIC ENTERITIS, BLOODY FLUX, DYSENTERY, ETC).

THIS disease is very common in the Avalon and surrounding districts, sometimes assumes the characteristics of a true epizootic, and affects young bovine animals between the ages of six months and two years, but is commonest and most contagious in animals of from ten to eighteen months old.

It attacks animals in good or bad condition, without distinction of breed or species. It begins towards the end of July, attains its maximum development towards the end of August and September, and disappears in October, though occasionally it continues until November. In exceptional cases Degoix has seen it during January and February in animals which had returned from the fields to the byres about the end of November. It developes earlier than verminous bronchitis, in conjunction with which, however, it often occurs. It is commonest in warm, moist, rainy years, and amongst animals pastured on swampy ground containing numerous springs and streams. Year after year it attacks animals occupying particular pastures in summer. The soil of these pastures is undoubtedly infested with the germs of the disease, just as in places the soil is infested with anthrax bacilli. The appearance of symptoms is preceded by an incubation period of one or two months. The length of this incubation period is fixed by the observations which Degoix has made during the past twenty years, and depends on the time which elapses between the animals being turned into infected pastures and the appearance of the preliminary symptoms.

The disease commences with liquid, serous, fœtid, greenish-black diarrhœa, the material being voided without special straining and the animals losing neither their spirits nor appetite. Fever can scarcely be detected, the temperature ranging between 38° and 39° centigrade. On the second or third day the diarrhœa changes in character. Though it always remains fœtid, it now becomes mucous, reddish-black, or sanguinolent, and contains more or less frequent blood clots of varying size.

The passage of this material causes violent straining, which becomes more and more common and is accompanied by very pronounced rectal tenesmus. The animals stand with their backs arched for one or two minutes, sometimes longer, and the liquid escapes in large quantities, soiling the quarters and hocks. The animals are dull, show a certain degree of colic, and frequently grind the teeth. Appetite is lost, thirst is severe, and rumination ceases. Wasting makes rapid strides, the coat stares, the animals have difficulty in standing on account of their weakness, fever sets in, and the temperature rises to 40° centigrade. This condition may last from five to ten days and terminate either in recovery or death. Recovery is frequently rapid in animals which have continued to eat, and in which the acute period has been of short

duration—five to seven days at most. On the other hand, it is slow if the appetite has disappeared and the acute period has been prolonged beyond ten days.

The diarrhœa, which has lost its sanguinolent character towards the sixth or eighth day, may continue for somewhat longer. The attacks of straining become rare and cease between the tenth and fifteenth days. The appetite remains capricious for a long time.

Convalescence is marked by alternate improvement and retrogression. The animals are weak, and only recover quickly under energetic treatment and forced feeding with concentrated digestible foods like milk, soup, cooked grain etc., administered for three weeks or more.

Death may occur towards the tenth or fifteenth day from exhaustion. The patients become very anæmic and thin, the eyes are withdrawn into the orbits, and the animals appear indifferent to what goes on about them. They still groan feebly, occasionally grind the teeth, and lie continually on the chest with the head extended. The body temperature falls and death follows.

In well bred animals in good condition the disease sometimes assumes a much graver and more rapidly progressive form, with peracute symptoms, and makes as many if not more victims than that previously described.

The process is as follows : After suffering for a day from serous diarrhœa, to which the owners pay little attention, the animals show sanguinolent diarrhœa and pass blood clots. This is almost immediately followed by very violent convulsive attacks—true eclampsia. The animals are then unable to stand, lie on the side with the head outstretched and resting on the ground, the eyes withdrawn into the sockets and often showing pirouetting movements (nystagmus), the neck drawn upwards and backwards (opisthotonos), and the limbs rigidly extended. From time to time the whole body is shaken by extremely violent convulsive movements.

This condition, which is sometimes preceded by weakness of the hind quarters and symptoms of locomotor ataxia and incoordination, may continue from six to thirty-six hours ; in nine cases out of ten it terminates in death.

On microscopic examination of the serous dejections one finds distributed throughout the liquid mass very small numbers of ovoid corpuscles having a double outline, and contents of varied appearance ; these are the coccidia.

When the diarrhœa has become sanguinolent and muco-fibrinous, the fluid contains these coccidia in considerable quantities, and large numbers of them may be found in the mucus, where they are mixed with epithelial debris, blood corpuscles, and lymphatic cells, etc. They are rarer in the clots. Coccidia cannot be found in the fæces of healthy animals, even in those occupying the same pastures with the diseased. Should the clinical symptoms be thought insufficient of themselves clearly to identify the disease, a simple microscopic examination of the fæces will remove any doubt.

Post-mortem examination immediately after death enables one exactly to identify the habitat of the parasite and the lesions it produces. These lesions are to be found throughout the large intestine, from the cæcum to the anus.

The large intestine is almost or entirely empty, the mucous membrane is reddish-brown in colour, lies in folds, is œdematous, and everywhere covered with a coating of mucus. This coating varies in character at different points ; in places it forms more or less thickened patches of greyish or yellowish colour, and of a resistant character, as though mixed with coagulated fibrin. These patches are fairly well defined, they are irregular in form and vary in width from some millimetres up to several centimetres. They are more or less adherent to the mucous membrane, from which they can easily be stripped away. The mucous membrane thus exposed is slightly compressed, of a whitish colour, thus markedly contrasting with the surrounding red coloration. This depression represents a slight ulceration, which though superficial is clearly visible to the naked eye.

Microscopic examination of the mucus patches reveals the existence, both superficially and in the depths, of epithelial cells derived from the mucous membrane, of vesicular cells derived from the Lieberkuhnian follicles, of numerous blood and lymphatic corpuscles, and, distributed irregularly throughout this mass of cells, of coccidia, resembling those found in the dejections.

In thin sections of the intestine, made through the ulcerated mucus-covered patches, and in a direction perpendicular to the mucous membrane, one finds that the epithelial covering of the intestine has disappeared.

The Lieberkuhn's follicles are shortened, their orifices are irregular and partly blocked with epithelial debris. In a large number of these follicles the blind extremity is dilated, and more or less filled with coccidia varying in appearance according to their stage of development. The epithelial cells normally lining these blind ends seem to have disappeared, and to have been replaced by the parasites. This however is not really the case. It is easy to prove by suitable dissection (after maceration of the sections in 30 per cent. alcohol) that the coccidia are lodged in epithelial cells which have become modified in shape and undergone hypertrophy as the parasite has grown; whilst the nucleus has been pushed to one end and undergone atrophy.

Alongside the diseased glands may be found others which are quite healthy. In the parts which are most markedly affected the interglandular connective tissue is infiltrated and slightly thickened. Degoix has never discovered coccidia in the cells of the intestinal mucous membrane itself. The lesions may be met with throughout the large intestine, but are more numerous and grave as the rectum is approached.

This disease is characterised by extensive inflammation, affecting the entire mucous membrane of the large intestine, by more or less extensive ulceration of this bowel, the ulcers being covered with muco-fibrinous patches rich in coccidia, by localisation of the parasites in the epithelial cells of the glandular cul-de-sac, by the appearances of the disease at a special time of year and in special districts, where it assumes the form of an enzoöty, and by the fact that it always appears in the same pastures. The mortality varies between 12 and 25 per cent. from year to year.

The protozoa which produce this disease assume the form of ovoid cysts, varying in length from 18 to 25 μ, and measuring at the widest part about 13 μ. They possess a hyaline envelope whose existence is proved by its double contour line, and yellowish granular highly refractile contents. This granular material does not always occupy the whole of the cavity. At a certain stage of development it collects towards the centre, forming a nucleus and leaving clear spaces at the poles of the cell. At a later stage this nucleus divides into four portions which afterwards separate.

The protozoa, in their cystic condition, are very resistant to destructive influences. The most common natural cause of their destruction is drying in the open air. Unfortunately, in the localities where the patients usually live, that is, filthy byres, wet pasturages, etc., this destruction only takes place to a very limited extent.

The disease can be attacked by prophylactic and therapeutic measures.

Prophylaxis consists firstly in destroying the parasites contained in the dejections by the free use of 3 per cent. sulphuric acid solution, and secondly in removing the young animals from infected fields during the months between June and September.

Therapeutic treatment comprises:—Firstly, stimulant applications to the abdomen. Secondly, disinfection of the intestine by the administration of salol, benzo-naphthol', very diluted solutions of creolin, etc. Thirdly, intestinal irrigation by the administration of mucilaginous drinks containing bi carbonate of soda, supplemented by general tonic treatment and the supply of concentrated, very nourishing, and easily digested food (Degoix. *Revue générale de Médecine Vétérinaire*, No. 28, 15th February 1904, p. 177).

To begin with, does ligature or section of the vas deferens react in any way on the testicle? Does this organ really retain its morphological integrity after such an operation?

This question MM. Ancel and Bouin answer in the negative. They point out that in 1898 they made a study of this question, and found that, after a longer or shorter period, spermatozoa were no longer formed, and that later still the spermatocytes and the spermatogonia themselves disappeared. In the case of a guinea-pig this occupied about 100 days; but in that of the rabbit the time must be considerably extended.

These experiments led them to conclude that the whole of the sexual gland disappears after the excretory ducts have been occluded.

But, if one studies the organs more closely, one finds, between the seminiferous tubes, a certain variety of cells which do not show any sign of degeneration under the above circumstances. They are the "interstitial" cells of the testicle, the function of which until now has remained obscure, and the great importance of which the two authors have sought to emphasise in a recent observation. They believe that it is owing to the special activity of these cells that all the results previously attributed to recrementitious secretion must be ascribed.

The morphological characters of the interstitial cells, their greater or less abundance in different species, their arrangement round the blood-vessels, their histological development, etc., have long been known. The two authors have recently shown that these cells possess all the characteristics of glandular cells, that they secrete actively, and that at least a portion of their secretion is poured into the blood-vessels and lymphatics.

The great majority of authors attribute to these interstitial cells a trophic action as regards the sexual elements, but in fact they appear to be quite independent of the seminal gland.

Apart from the morphological distinctions established by experiment and by the pathological conditions above referred to, a great many other arguments may be advanced in favour of this view.

The two most important refer to the method of development of the internal cells in cases of ontogenesis of the testicle and in the testicles of cryptorchid animals. These cells become considerably developed, and are functionally active from the time when the genital tract is first differentiated. Their function is therefore developed very early (in pig embryos of 30 mm. in length), whilst the vas deferens is and for long remains in an embryonic condition, and is undergoing practically no developmental change. Nothing similar can be detected in the female. On the other hand, retained testicles show still more clearly the same morphological dissociation. In the great majority of cases the seminiferous tubules of these organs do not contain and never have contained the different cells from which spermatozoa are finally evolved, but the interstitial cells interposed between these sterile tubules, are quite normally developed in all cases. They have been evolved normally whilst growth of the seminiferous tubules has been arrested.

The above-mentioned facts led the authors to conclude that the interstitial cells together form a glandular organ which is relatively independent of the process of forming spermatozoa, and which performs a useful function.

They think this function is extremely complex, and that from the earliest period of embryonic life it probably plays an important part in development and growth. They are also of opinion that the interstitial gland of the testicle performs among other physiological functions that of determining the secondary sexual characters of the male and the sexual instinct. They entirely reject the theory of the recrementitious function of the testicle (Ancel and Bouin. *Receuil de Méd. Vétérin.*, 15th January 1904, p. 78).

IMMUNISATION AGAINST ANTHRAX BY THE USE OF VIRULENT CULTURES.[1]

By J. A. GILRUTH, M.R.C.V.S., Chief Veterinarian, Department of Agriculture and Pathological Public Health Department, New Zealand.

THE experiments which I propose to describe arose through the observation of peculiar symptoms exhibited by a guinea-pig after inoculation with a small quantity of blood from an animal dead of anthrax, this blood having been accidentally contaminated by another organism during collection.

As is well known, a guinea-pig or a rabbit inoculated with even a small quantity of a pure culture of anthrax bacilli succumbs within twenty-four to forty-eight hours, and then with comparatively no premonitory symptoms, while no evidence of inflammatory action is exhibited at the seat of inoculation. In this case, however, there was around the region of inoculation for the first two days intense inflammation, which nevertheless produced practically no general disturbance, and rapidly disappeared almost entirely. On the fifth day after inoculation the animal was, so far as could be observed, in the best of health, yet on the morning of the sixth day (exactly 140 hours after inoculation) death occurred.

Post-mortem examination disclosed a typical anthrax condition, with subcutaneous gelatinous œdema and enlarged spleen; while microscopical examination demonstrated enormous numbers of bacillus anthracis in the spleen and other organs.

[1] A Paper read before the Annual Meeting, New Zealand branch of the British Medical Association, at Wellington, on 16th March 1904.

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It is interesting to note that a pure culture of the bacillus anthracis had been separated from the original pipette of blood the day following the inoculation of the guinea-pig, and that another guinea-pig inoculated subcutaneously with a small quantity of the culture by means of the platinum needle succumbed in thirty hours, or three days before the one first inoculated. Further, two days after inoculation the œdema of the intense swelling at the seat of inoculation of the first animal contained *only* the accidental bacillus so far as could be demonstrated by microscopical and by cultural methods, proving that, at the least, development of the anthrax bacilli was being inhibited as a result, indirectly at all events, of the other's presence.

Inoculation of a quantity of pure culture of the accidental bacillus alone produced but a slight swelling of a very temporary nature at the seat of inoculation.

The anthrax cultures have always been virulent, as frequently proved by control experiments, rabbits and guinea-pigs inoculated with from one-twentieth to one-five-hundredth cc. of pure cultures in broth being sufficient to cause death within thirty-six hours, or at most under forty-eight hours.

A loopful of an agar culture of anthrax bacilli, inserted into a pocket underneath the skin of the thigh of a guinea-pig, and followed by a loopful of agar culture of the non-pathogenic organism found as a contamination previously mentioned (and which I shall term Bacillus X), produced only a faint swelling, the animal remaining normal otherwise.

One rabbit received subcutaneously one-twentieth cc.[1] anthrax culture plus one-twentieth cc. bacillus X[2] (broth cultures), and remained normal; while another showed for four days but a slight local swelling after being inoculated with similar quantities, but death of typical anthrax resulted on the fifth day after inoculation. At *post-mortem* examination only the bacillus anthracis could be demonstrated. This induced me afterwards to generally give double the dose of the " foreign " bacillus to that of anthrax.

Rabbits inoculated with mixtures consisting of one-twentieth cc. bacillus anthracis plus one-twentieth cc. bacillus X, and with one-tenth cc. bacillus anthracis plus one-fifth cc. bacillus X, remained normal.

One rabbit received one-tenth cc. of anthrax mixed with double the dose of bacillus X, and, remaining normal, was inoculated nineteen days later with one-twentieth cc. each of anthrax and X cultures. The second inoculation producing no ill effects, eight days later one-twentieth cc. of anthrax alone was injected, with the result that death of typical anthrax occurred about fifty-six hours later, indicating that probably some degree of resistance had been conferred by the previous inoculations, especially as another rabbit not previously treated died within thirty-six hours after inoculation with the same dose of anthrax bacilli.

A rabbit inoculated in one thigh with one-twentieth cc. of bacillus anthracis, and in the other thigh with one-tenth cc. of bacillus X,

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[1] Cultures used were twenty-four hours old broth cultures.

[2] The chief characteristics of the bacillus are as follows : Short, varying from almost a coccus to almost the length of B. typh. Not motile. Does not stain by Gram's method. Grows readily in gelatine, forming in two days a definite growth with irregular edges and a faint bluish tint. On agar forms a thick growth in eighteen to twenty-four hours at 37° C. It does not grow on potato, does not coagulate milk, and forms gradually a thick scum on the surface of broth, which it renders very cloudy.

died within thirty-six hours of typical anthrax, showing that the organisms must be mixed before any resistance to the anthrax germs could be exhibited by the animal.

Experiments with Bacillus Coli Communis.

The bacillus coli used had been cultivated for many generations, and produced a slight local inflammatory reaction when injected alone.

A young rabbit was inoculated with one-twentieth cc. anthrax and one-tenth cc. coli bacilli. Death resulted in thirty-six hours. Autopsy showed much swelling with hæmorrhage at the seat of inoculation, and separation of the muscles by sanguineous œdema. The joints, particularly the carpal and tarsal, were inflamed and surrounded by hæmorrhagic œdema. From the subcutaneous œdema both the organisms injected were isolated, and the spleen was found to contain both anthrax and coli bacilli in numbers. The subcutaneous tarsal and carpal lesions were probably due to the colon bacillus alone.

Another rabbit was inoculated with one-tenth cc. anthrax and one-fifth cc. coli bacilli. Some inflammation and œdema developed at the seat of inoculation for the first few days, the general health remaining fairly normal. On the morning of the fifth day death resulted, *post-mortem* examination revealing a typical anthrax condition, and only the bacillus anthracis could be recovered.

Another rabbit was inoculated with one-tenth cc. anthrax bacilli and three times the quantity of bacillus coli. Swelling and œdema developed at the seat of inoculation by the second day, but the animal otherwise was normal. Death occurred on the morning of the fifth day of typical anthrax, and only the bacillus anthracis could be recovered on autopsy.

Experiments with the Streptococcus.

A rabbit inoculated with one - twentieth cc. anthrax plus one-twentieth cc. streptococcus (isolated from a diphtheritic throat) developed a slight inflammatory swelling at the seat of inoculation, but otherwise remained apparently normal till found dead on the morning of the sixth day. Autopsy disclosed a small collection of pus at the seat of inoculation and a small purulent nodule in the precrural gland. Streptococci and anthrax bacilli were found in the pus, but in the spleen, blood, etc., only anthrax bacilli could be demonstrated.

Another rabbit inoculated with one-tenth cc. anthrax plus one-fifth cc. of another culture of a streptococcus (isolated from a case of human septicæmia) developed a slight inflammatory swelling at the seat of inoculation, and in two days pus was present, though otherwise the animal remained normal. On the fourth day death occurred. Autopsy disclosed a quantity of thick pus at the seat of inoculation, which under the microscope appeared to contain only streptococci. The spleen and blood were typical of anthrax, but were found to contain both organisms in numbers.

It is interesting to note that neither of the above streptococci was pathogenic for guinea-pigs or rabbits when injected alone, a rabbit which received even one-fourth cc. of the latter developing no abscess.

Experiments with Bacillus Enteritidis (Gaertner).

The original culture of this bacillus which I received was brought from the London Hospital by Dr Makgill, District Health Officer, Auckland. It has been cultivated in my laboratory for many generations, and now possesses no virulence, at least for sheep and rabbits, when injected alone.

A large number of experiments have been conducted with a mixture of this bacillus and that of anthrax, and practically all attempts at conferring immunity have been made by injecting the anthrax bacillus with this bacillus, chiefly because it is a well-known variety.

Full-grown rabbits have received up to one-half cc. of anthrax culture with impunity as a first dose when mixed with double the quantity of enteritidis culture.

The first sheep inoculated with the mixture received one-twentieth cc. of anthrax plus one-tenth cc. of enteritidis cultures and remained normal till the eighth day, when death occurred, *post-mortem* and bacteriological examinations demonstrating typical anthrax conditions. This animal was, however, about four days after inoculation, shorn owing to the hot weather, which immediately afterwards changed to bitterly cold, and it is probable that this had a definite effect in weakening the animal's resistance. This explanation is rendered all the more probable by the fact that later a full-grown ewe received a similar dose of anthrax and enteritidis bacilli, while a lamb received as a first injection the enormous dose of one-half cc. of anthrax culture plus 1 cc. enteritidis, neither developing any symptoms of sickness, beyond the fact that the lamb evinced slight lameness of the inoculated leg during the day following the inoculation.

A pig about three months old received subcutaneously one-quarter cc. of anthrax with double the quantity of enteritidis bacilli, and showed no ill effect. Eleven days later the animal accidentally was allowed to partake of a small quantity of blood and flesh of another animal dead of anthrax, and, beyond showing slight langour with rise of temperature the following day, no ill effects ensued. It is possible, therefore, that some immunity had been conferred by the first inoculation, though it must be borne in mind that swine are not readily affected by anthrax per os. At all events, twenty-two days after the animal had fed on anthrax material another injection of 1 cc. anthrax culture with 2 cc. enteritidis was borne with impunity.

It should be noted that even when death due to anthrax was delayed much beyond the normal time, as in the cases when the bacillus was mixed with coli and with the streptococcus, and in the sheep which died of anthrax eight days after being inoculated with anthrax plus enteritidis, no diminution of the virulence of the recovered anthrax bacilli has been observed.

Immunity Conferred.

A rabbit (136) was inoculated with (1) one-tenth cc. of anthrax culture; (2) nineteen days later, one-quarter cc. anthrax culture; (3) fifteen days later, one-half cc. anthrax culture; (4) eighteen days later, 1 cc. of anthrax culture; in each case the anthrax being mixed with double the quantity of enteritidis culture. As none of the inocu-

lations produced any deleterious effects, (5) one-twentieth cc. of anthrax alone was injected fourteen days later to ascertain if immunity to that dose had been produced. The animal remained normal, proving the acquisition of a very considerable immunity. (6) Fifteen days later, one-tenth cc. anthrax culture alone; (7) fourteen days later, one-quarter cc. anthrax culture alone; (8) eighteen days later, one-quarter cc. anthrax culture alone; (9) twenty-four days later, one-quarter cc. anthrax culture alone; (10) fifteen days later, one-half cc. anthrax culture alone; (11) fourteen days later, three-quarters cc. anthrax culture alone; (12) sixteen days later, 1 cc. anthrax culture alone were administered; and none of these inoculations was followed by any bad results, proving that this animal (exhibited) has acquired an enormous degree of immunity. Altogether, within a period of six months, this animal has received 5 cc. of anthrax broth culture subcutaneously, half of the quantity being injected in a state of purity within a period of nine weeks.

Another rabbit (141) was inoculated with (1) one-twentieth cc. anthrax culture plus four times the dose of enteritidis; (2) twenty days later, one-half cc. of anthrax plus 1 cc. of enteritidis cultures was injected. As after each inoculation the animal remained normal, (3) twelve days later one-twentieth cc. of anthrax culture alone was injected with negative results. (4) Fifteen days later, one-tenth cc. of anthrax culture alone; (5) twenty-four days later, one-tenth cc. of anthrax culture alone; (6) fifteen days later, one-quarter cc. of anthrax culture alone; (7) fourteen days later, one-half cc. of anthrax culture alone; (8) sixteen days later, one-half cc. of anthrax alone were administered subcutaneously; and none of these inoculations was followed by any bad results, proving that great immunity could be conferred fairly rapidly through injection of the double cultures.

Another rabbit (151) was inoculated with (1) one-tenth cc. anthrax culture plus one-fifth cc. of enteritidis; (2) fifteen days later with 1 cc. anthrax plus 2 cc. enteritidis. As no pathological effects were observed to follow the second injection of such a large dose of both organisms, (3) fourteen days later the large dose of 0·15 cc. of anthrax alone was injected. The result showed that not sufficient immunity to combat this dose had been acquired, for the animal died about forty hours afterwards of typical anthrax. At the seat of the second inoculation a considerable quantity of greyish stiff purulent material was present, with necrosis of portions of the muscles of the groin, probably due to the large quantity of enteritidis injected at the second inoculation, though none of these bacilli could be demonstrated by microscopical or bacteriological examination of the pus.

The ewe previously mentioned received (1) one-twentieth cc. anthrax culture; (2) eleven days later, one-half cc. of anthrax culture; (3) fifteen days later, 1 cc. of anthrax culture; (4) thirty-three days later, 5 cc. of anthrax culture; in each case combined with double the quantity of enteritidis culture. After the first three inoculations no bad effects were observed, but after the fourth the animal developed a hard circumscribed swelling at the seat of inoculation. This swelling attained its maximum on the second day, was flattened, and about 3 by 4 inches in measurement. It rapidly decreased in size by the fourth day, only a small indurated nodule remaining. There was slight lameness at first, but no pain or œde-

matous swelling. (5) Fifteen days after the last inoculation the sheep was inoculated with one-half cc. anthrax alone, and, proving immune, (6) twenty-one days later with 1 cc. anthrax alone. After the last inoculation slight lameness was evinced and a small swelling developed, both of which rapidly disappeared. This animal had therefore acquired a large degree of immunity. In a little over three months this sheep had been inoculated with over 8 cc. of virulent anthrax cultures, and had within three weeks withstood 1½ cc. of anthrax culture injected in a pure state with comparative immunity.

The lamb previously mentioned received (1) one-half cc. anthrax mixed with 1 cc. of enteritidis. As the animal remained healthy, (2) sixteen days later one-twentieth cc. of anthrax culture alone was injected, and to this the animal proved to be immune.

In considering these experiments it should be remembered that, in equal quantities of broth cultures of such organisms as bacillus enteritidis (Gaertner), bacillus coli, bacillus X, the streptococcus, and bacillus anthracis, there are many more organisms per cc. in the first three than in the last two, so that in this paper I must be understood to deal with quantity of cultures, not number of organisms.

Control experiments have proved that the cultures of bacillus anthracis used have always been capable of killing a rabbit or guinea-pig in less than thirty-six hours when used alone; and from the results obtained by mixing the bacillus anthracis with other organisms, as related, the following conclusions can, I think, be drawn :—

(1) Guinea-pigs, rabbits, and sheep can completely resist the inoculation of large doses of virulent anthrax bacilli, provided these organisms are mixed with a larger quantity of certain other organisms which are non-pathogenic for these animals.

(2) Death is frequently delayed very considerably when the organism mixed with the anthrax bacillus is possessed of slight pathogenic properties.

(3) The anthrax bacillus must be *mixed* with the other organism, for if they be injected under different parts of the skin no resistance results.

(4) An animal which has suffered with absolute impunity the injection of a large dose of anthrax bacilli when mixed with a foreign organism may succumb later to a much smaller dose of pure anthrax culture, although generally some resistance is evidenced (*see* Rabbit 151).

(5) In spite of this, however, it has been observed with rabbits and sheep which received repeated doses of both anthrax and Gaertner's bacilli in increasing quantities, that immunity to large doses of pure anthrax cultures could be conferred.

(6) And if this phenomenon be further investigated, it may possibly afford a surer and more satisfactory method of conferring immunity against anthrax, and possibly other diseases, than those now in vogue.

That these animals have no natural immunity to anthrax is well known. How comes it, then, that the system can repel the invasion of such a number of virulent anthrax bacilli simply because they are accompanied by organisms which to all intents have no pathological significance?

In order to approach this question satisfactorily one must consider

the different effects of the introduction of the bacillus anthracis into the human and the lower animal.

As is well known, the introduction of bacillus anthracis into the human system through an abrasion of the skin produces an intense local reaction, known clinically as a malignant pustule. In other words, a fierce battle takes place between certain of the white blood cells, which crowd in enormous numbers to the spot, and the invading bacilli. In this battle many of the individuals on both sides perish, but, as a result of this opposition, cutaneous inoculation with anthrax bacilli in man, provided surgical treatment is available in time, and the initial dose of the bacilli has not been too great, is not often attended with fatal results.

Inoculation with the anthrax bacillus in the lower animal, however, is followed by no such warfare. As a rule even twenty-four hours after inoculation the seat presents a normal appearance, there being no inflammatory changes evident. Death is almost appalling in its suddenness. Frequently I have observed experimental animals half-an-hour before death in apparent perfect health. Often even on *post-mortem* examination there is no lesion to be observed near the seat of inoculation, though generally there is a greater or less amount of clear œdematous fluid in the vicinity. The notable point in regard to the œdema is that it is almost totally devoid of the white blood corpuscles, particularly those which have been proved to have a bactericidal action. In other words, whereas in man the protective cells of the body fight every inch of the ground with the invading bacilli, in the lower animal a free field is left, there not being even a faint local inflammatory reaction.

My experiments, however, prove that when this bacillus is mixed with an organism that alone would cause a local congestion or inflammation, the latter does not lose this property, and that consequently the protective cells are brought to the spot. In the resultant fight both the "foreign" organism and the anthrax germ are attacked by the phagocytes, and although the anthrax germ ultimately prevails, it is only after the lapse of a much greater time than occurs under ordinary experimental conditions.

In the other cases, where practically no swelling or congestion occurs, and where the virulence of the anthrax bacilli is most completely baffled (such as the rabbit which received subcutaneously with immunity 0·5 cc. of the bacillus when mixed with double the quantity of bacillus enteritidis), the circumstance is perhaps more difficult of explanation. This is a point which in itself would require to be the subject of extended investigation.

It seems probable that the X bacillus and Gaertner's bacillus, which possessed no pathogenic power alone, being an easy prey to the phagocytes, and having no harmful effect upon these cells, were rapidly conquered soon after inoculation, and along with them the anthrax bacilli, before they had time to multiply. But these points will repay much more attention than I have had time or opportunity to bestow upon them as yet.

The comparative rarity with which animals are affected with anthrax, even when grazing continually upon lands badly infected with anthrax germs, has been the subject of frequent remark. The experiments which I detail in this paper, however, offer a satisfactory

reason.   No doubt, in districts where anthrax is indigenous, the bacillus frequently gains entrance to the system, but, as must be the case under most circumstances, being accompanied by numbers of other organisms of a benign nature, no deleterious results ensue unless either the dose of anthrax bacilli is very great or the dose of the other organisms is very small.

Tables showing Inoculations of Anthrax Virulent Bacilli employed in conferring of Immunity.

RABBIT (136).

Inoculations.	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·10 cc.	0·20 cc.
(2) 19 days later	0·25 „	0·50 „
(3) 15 „ „	0·50 „	1·00 „
(4) 18 „ „	1·00 „	2·00 „
(5) 14 „ „	0·05 „	None.
(6) 15 „ „	0·10 „	„
(7) 14 „ „	0·25 „	„
(8) 18 „ „	0·25 „	„
(9) 24 „ „	0·25 „	„
(10) 15 „ „	0·5 „	„
(11) 14 „ „	0·75 „	„
(12) 16 „ „	1·00 „	„

RABBIT (141).

Inoculations.	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·05 cc.	0·2 cc.
(2) 20 days later	0·50 „	1·00 „
(3) 12 „ „	0·05 „	None.
(4) 15 „ „	0·10 „	„
(5) 24 „ „	0·10 „	„
(6) 15 „ „	0·25 „	„
(7) 14 „ „	0·50 „	„
(8) 16 „ „	0·50 „	„

RABBIT (151).

Inoculations.	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·1 cc.	0·2 cc.
(2) 15 days later	1·00 „	2·00 „
(3) 14 „ „	0·15 „	None.

Death occurred in forty hours, showing that not sufficient immunity for the third dose had been acquired.

EWE.

Inoculations.	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·05 cc.	0·1 cc.
(2) 11 days later	0·5 „	1 „
(3) 15 „ „	1 „	2 „
(4) 23 „ „	5 „	10 „
(5) 15 „ „	0·5 „	Alone.
(6) 21 „ „	1 „	„

SHEEP G (*Lamb, 6 months*).

Inoculations.	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·5 cc.	1·00 cc.
(2) 16 days later	0·05 „	None.

PIG.

	Dose of Anthrax Bacilli.	Dose of B. Enteritidis.
(1)	0·25 cc.	0·5 cc.
(2) 10 days later, fed accidentally on quantity of blood swarming with anthrax bacilli from dead cow. Slight languor following day. Rapid recovery.		
(3) 22 days later	1·00 cc.	2·00 cc.

AURICULAR ACARIASIS.

By FRANK GARNETT, M.R.C.V.S., Windermere.

THE classification of the order acarina, though far from being finally settled in regard to the position which some of its families may hold, is sufficiently clear as regards the position of the sarcoptidæ. The magnitude of the work of classifying this huge order has been attempted by many naturalists during the past century. Linnæus in 1735 treated it as one genus, but De Geer may be said to have been the first, in 1778, to attempt a classification, following whom we have Latreille and Herman in 1806, C. Von Hayden in 1828, Duges (1839), Kock (1842), Nicolet (1855), Früstenberg (1861), Donadieu (1875), Magnin (1876), Kramar (1877), Michael (1884), and in 1891 Canestrini of Padua and Trouessart of Paris.

Each succeeding acarologist since De Geer's first classification has differed from his predecessors, some dividing the order according to a single organ, as palpi; form, as vermiform and non-vermiform; habitat, as terrestrial and aquatic; till P. Kramar of Schlensingen divided the order by the tracheæ into tracheata and atracheata, which main divisions have been generally accepted till the classifications of 1891 appeared.

Canestrini (1891) treats the acari as a class, and divides it into six orders, the first of which is astigmata, which is divided into two sub-orders (1) vermiforma, (2) sarcoptina; the former is divided into two families, of which one is the demodicidæ, the latter into seven families, the second of which, psoroptidæ, contains three genera, sarcoptes, psoroptes, and symbiotes. In Dr Trouessart's 1891 classification the mites are divided into two orders, acarina and vermiformia, the latter containing the family demodicidæ. The acarina he divides into three sub-orders, prostigmata, metastigmata, and astigmata, the last containing a single family, sarcoptidæ, which he divides into six sub-families, included in which is the sarcoptinæ.

It is to the astigmata or atracheata (Kramar), without tracheæ and

therefore without stigmata, that the family sarcoptidæ belong, the respiration being entirely cutaneous.   The acari belonging to this family may be distinguished by having in the adult four pairs of legs of five articles each, attached to a soft body by chitinous epimera ; the mandibles are chelate, and there are no eyes.

The sarcoptidæ is a very large family, and is divided into five to seven sub-families, of which one, sarcoptidæ psorica or sarcoptinæ, contains all the acari parasitic to animals and causing the disease known as scabies, with the exception of demodectic scabies, the parasite of which belongs to the family demodicidæ.

The sub-family sarcoptinæ is divided into three distinct genera, sarcoptes, psoroptes, and symbiotes, each supplying one or more species producing a distinct form of scabies.

The sarcoptes are divided into two species, sarcoptes scabiei and sarcoptes minor, the former attacking horses, dogs, cattle, and pigs, while the latter are peculiar to cats and rabbits.   The form of mange caused by either of these species is the most troublesome to eradicate on account of the burrowing proclivities of the ovigerous female. The genus psoroptes contains a single species, psoroptes communis, and it is the variety ovis of this species which is the cause of the disease known in this country as sheep scab.   The species also attacks horses, cattle, and rabbits.

The genus symbiotes contains four species, two of which are known respectively as symbiotes communis and symbiotes auricularum. The former produces by far the commonest form of parasitic affection among horses, and the latter species is a very frequent cause of auricular disease in dogs and cats.

The following classification of parasitic acari producing scabies in the domestic animals would, I think, be accepted as correct by all naturalists at the present time.

Order.	Sub-order.	Family.	Sub-family.	Genera.	Species.
Acarina	Astigmata or Astracheata	Sarcoptidæ	Sarcoptidæ psorica or Sarcoptineæ	Sarcoptes	Sarcoptes scabiei. Sarcoptes minor.
				Psoroptes	Psoroptes communis.
				Symbiotes	Symbiotes communis. Symbiotes auricularum.

Symbiotes auricularum as a cause of otitis in dogs and cats has received but little attention at the hands of English veterinarians, and, with the exception of Mr A. J. Sewell in 1891, in the *Veterinary Journal* of that year, I can find no recorded cases, yet it is undoubtedly one of the commonest causes of that common disease in both these animals which we are called upon to treat.   Mr Sewell, in the communication referred to, calls the parasite psoroptes auricularis canis, but his illustrations, incorrect though they are in certain details, are sufficiently well drawn for anyone who is acquainted with the two species to say that it is not a psoropt but a symbiot, and, further, that it is the parasite now known as symbiotes auricularum, though in his description he mistakes the ova for larval forms, the adult

female for a male, and the pubescent female for a female, while the male is not figured at all.

It is usual to speak of symbiotes auricularum var. canis and var. cati, respectively, when the parasite is found in the ear of the dog or cat as the case may be. I am of opinion that they should not be treated as separate varieties, as I do not consider that habitat alone is of itself sufficient to make a distinct variety, the parasite being identical

FIG. 1.

Dry slip preparation of psoroptes communis var. ovis (×50). 1. Ovigerous females with ova. 2. Pair coupled. 3. Male. 4 and 5. Pubescent females. 6. Nympha. 7. Hexapod larva. 8 Ovum.

in every other respect. I have not been able to distinguish any external anatomical difference in the acari obtained from these two animals, though Neumann gives the size of the auricular symbiot of the dog as being larger than that of the cat.

The following case inclines me to the opinion that the parasite is intercommunicable from dog to cat. I treated a collie dog, which lived luxuriously in the house, for canker in the ear due to symbiotes auricularum. I ceased treating it before cure was complete, as the

owner thought it had so far recovered that it would go on without further attention. In the house was a cat of a wild disposition which could not be induced to go near the dog. Soon after I had given up treating the dog the owner had two kittens given from different homes. They became very friendly with the dog, all three constantly lying together before the fire or in the sun. About eight months after I first treated the dog it was brought again suffering from the disease in a more aggravated form, and I was also requested to go

FIG. 2.

Symbiotes auricularum, slip preparation (×50). 1. Ovigerous females. 2. Pair coupled.
3. Male. 4. Pubescent female. 5. Hexapod stage. 6. Ovum.

and see the two cats, which were continuously scratching and shaking their heads, the smell from them being very offensive in the hot close rooms. I found them both suffering from symbiotes auricularum, which were indistinguishable in all their stages of development from the acari obtained from the dog. The wild cat remained free from the disease.

The symptoms of auricular acariasis are peculiarly local, and are not noticed, as a rule, until the disease is considerably advanced, when the patient evinces irritation of the ears, manifested by holding the head on one side and constantly pawing and shaking them. An

examination reveals the internal parts of the ear to be highly in-
flamed, and a quantity of dark brown cerumen more or less fills the
internal concha ; this cerumen is hard and dry on the surface, but
·underneath it is soft and moist and of a light yellowish colour, and
in consistency is not unlike the peculiar sticky mucous-like exudate
which is seen in sheep scab in those situations where the psoropt
abounds.   The smell given off from the ear is very offensive.   In
advanced cases the lining membrane of the ear becomes thickened
and crenated to such an extent that the canal is practically closed.   I
have not seen any of the epileptiform symptoms which are described
by Continental acarologists.

If some of the cerumen and serosity are removed from the affected
ear (for this purpose I use a small thin bone ear-scoop) and examined
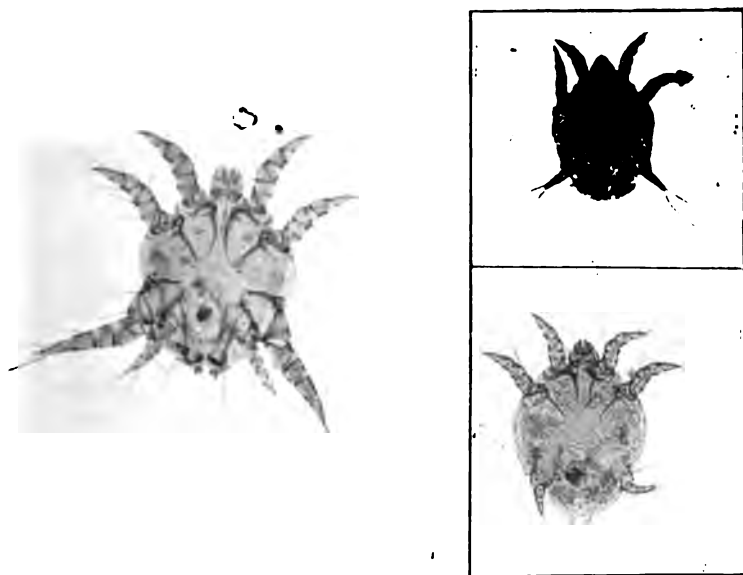
Fig. 3.
Symbiotes auricularum (×70). 1. Hexapod larva. 2. Pubescent female. 4. Male,
copulatory suckers extended.

under a hand lens, numbers of small white specks will be seen moving
about, their movements being comparatively rapid ; these are the
symbiots, and, if the cerumen which has been removed be placed on a
sheet of black paper for half an hour or so, the parasites will be found
gradually to congregate together in little masses containing ten to
twenty individuals or more.   Sometimes even in the ear similar
masses may be found and lifted off all together.   If further examina-
tion is desired, the cerumen should be placed on a glass slip with a
drop of clove oil upon it, and a cover glass pressed down and the
preparation examined under a low power of the microscope, Zeiss
a.a. or A being amply sufficient for the purposes of identification.   The
parasites will be seen in all stages of development from ova to adults,
with many pairs in coition.   The great disparity in the number of
males to females which is alluded to by authorities on this subject is

one which applies to all forms of psoric acariasis, and is not correct without considerable qualification. It depends (1) on the locality from which a given number of parasites are obtained, and (2) on the males being counted and compared with the number of ovigerous and pubescent females combined. (1) It will be found that there are more males and pubescent females together with larval and nymphal forms in the most recently invaded areas, and that the ovigerous females are more numerous towards the periphery of the scab, their
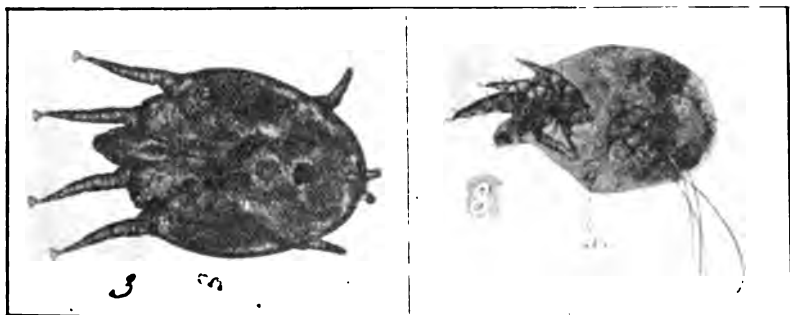
FIG. 4.

Symbiotes auricularum.   3. Pubescent female (×100).   7. Male (×70).
8. Ovigerous female, side view, anterior legs raised (×70).

function in this stage being entirely devoted to depositing their ova. (2) If the number of males be compared with the number of pubescent females, the ovigerous females being disregarded, it will be found that the number of males is little, if any, less than that of the pubescent females, i.e., females in an unfecundated condition and prior to their final ecdysis.

The disease yields to any parasiticide treatment which is efficiently carried out, provided the agent used is of sufficient strength to kill the parasites and yet not strong enough to increase the irritation in the ears.

Symbiotes auricularum are oviparous acari, differing somewhat from other species of sarcoptinæ in their life history. The development of an acarus is divided into three stages, ovum, larva, and nympha. To these must be added a fourth in the females of sarcoptinæ, which is known as the pubescent female stage. The term larva is applied to the recently-hatched acarus while it remains in a hexapod condition ; the term nympha is reserved for the next stage, when it has become octopod but without external evidence of generative organs. I am
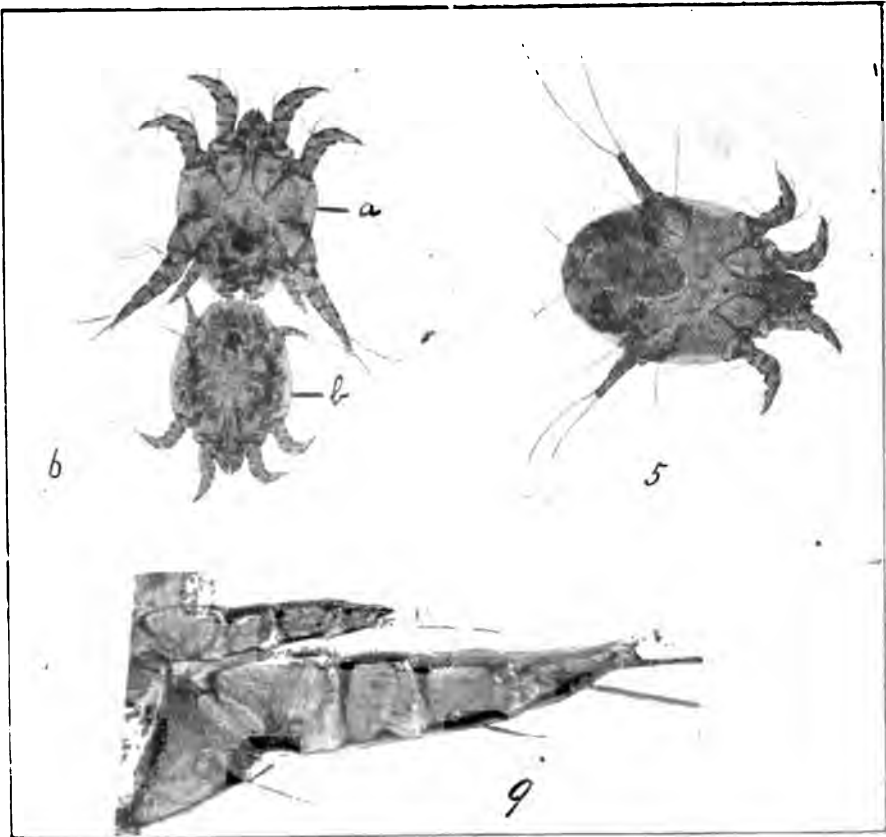
FIG. 5.

Symbiotes auricularum. 5. Ovigerous female with ovum (×70). 6. Pair coupled—a, male ; b, pubescent female (×70). 9. Left third and fourth legs of male (×300).

not aware that the development of the sarcoptinæ has been thoroughly made out, but in other sub-families of the sarcoptidæ closely allied to it, as tyroglyphinæ, where observations can be more easily made, the acari pass through a single ecdysis in the larval stage and two in the nymphal, which brings them to the imago ; but in addition to these in sarcoptinæ there is certainly another ecdysis in the female, which is the change from the pubescent to the ovigerous state.

The ova of symbiotes auricularum are small oval bodies, measuring ·21 mm. long by ·11 mm. wide ; they are of a white or greyish colour,

and not more than one is seen in the oviduct at a time. They are very large compared with the size of the acarus. Through the transparent capsule of the ova, prior to hatching, the doubled-up larva may be clearly seen, the two anterior pairs of legs folded backwards and the posterior ones forwards towards the rostrum. The colour of the hexapod larva on emerging from the ovum differs but little from that of the imago ; the abdomen is opaquish-white, the chitinised parts of the exoskeliton being of a pale yellow, which becomes darker in each succeeding stage of development. The two anterior pairs of legs in the larva, as in all the other stages, terminate with a small claw and a pedunculated caruncle or sucker ; the third or posterior pair in two small hairs. Excepting for an increase in size, there is in symbiotes auricularum nothing to distinguish the nympha from the larva, and, following the definitions of the stage as applied to acari, the nymphal stage must be looked upon as being entirely suppressed ; there is no appearance of a fourth pair of legs, and this applies also to the pubescent females, which can only be distinguished from the larva by the external generative organs, the copulatory tubercles, which are so highly developed and form such a striking feature in this species Neumann says that the fourth pair of legs of the pubescent female are reduced to knobs carrying a small hair. I have not been able to verify this in any of the large number of specimens I have examined, and have come to the conclusion that the pubescent female of symbiotes auricularum is still hexapod. The so-called vulvo-anal slit of the pubescent female is not functionally a cloaca, but only the external opening of the rectum. As long ago as 1861, Gudden discovered what is now known as the bursa copulatrix, which was rediscovered by Michael in 1879, and confirmed by many other acarologists since. In sarcoptidæ the female is fertilised through the bursa copulatrix, a small retroanally placed opening which leads to the receptaculum seminis ; this bifurcates to the ovaries and oviducts, which reunite immediately supero-posteriorly to the sub-thoracic vulva, through which the eggs are laid.

The final ecdysis of the male brings it to the pubescent or imago state, in which it differs from the other stages by the greatly developed third and fourth pair of legs. The fourth pair are larger and stronger than in any other species of sarcoptinæ, and the caruncle on the tarsus is equal in size to those on the other legs. The third pair of legs presents a distinctly peculiar ambulacrum (Fig. 9, 5), the tarsus being terminated by a truncated sucker in addition to the pedunculated caruncle which arises from its inner surface. The posterior lobes of the male are slightly-rounded projections carrying three hairs each, the centre one being the longest ; the lobes are separated by a small notch. The copulatory suckers are situated somewhat forward from the posterior margin of the body, and are protected by roundish chitinous pieces. The average size of the male is ·36 mm. long by ·26 mm. wide.

Coitus persists for a long time between the pubescent female and male in symbiotes auricularum, as is common with other species of sarcoptinæ, the coupling being brought about by the suckers of the male extending a fine membraneous sheath which envelopes the tubercles of the female, the ends of which are held by the powerful suckers of the male. It is during coition that the final ecdysis of the

female takes place, and I believe that act is not complete till the fecundated female emerges from the exuvium.

The average size of the ovigerous female is ·44 mm. long by ·3 mm. wide. The third pair of legs in this stage are large, and terminate with two long hairs, the fourth pair of legs being rudimentary, the tarsus barely extending beyond the body and ending in two small fine hairs. The vulva is situated centrally between the posterior end of the epimera of the second and the anterior end of the epimera of the third pair of legs, the crenated appearance of the derma, with the supporting sclerates converging to the opening, being a striking feature on the ventral surface of the body.

The inadequate anatomical descriptions of symbiotes auricularum which are available, and the difference of those which are given from the ones I describe above, leave two conclusions open—either the species I describe is a new one, or the differences are due to lack of description in the previous literature. I am inclined to believe that the latter is the right explanation, though the illustrations of the male given by Neumann and his description of the pubescent female differ from mine very considerably.

In order to convey an idea of the size of symbiotes auricularum, I have given photomicrographs of that parasite and of the common psoropt which is so well known, under the same magnification.

TICKS AND MALIGNANT JAUNDICE OF THE DOG.[1]

By CHAS. P. LOUNSBURY, B.SC., F.E.S., Government Entomologist, Cape Town.

MALIGNANT jaundice is the dog disease commonly known in South Africa as distemper, or hondziekte. The name here adopted is the one preferred and long used by the Cape Veterinary Department; the terms malignant malaria and bilious fever have also been applied to the disease by veterinarians. As the most serious, and one of the most common diseases affecting dogs in the country, its own importance warranted the attention I have given to its means of transmission from animal to animal; but the real inducement to study its means of dissemination was the hope of acquiring knowledge that might prove of value in later investigating diseases of other animals that are of greater economic consequence. Malignant jaundice is one of a large number of diseases of man and lower animals due to the presence and multiplication of protozoan organisms in the blood; and these diseases, with others of more obscure nature, as, for instance, heartwater, horse-sickness, and yellow fever, are probably all dependent on blood-sucking parasites for their transmission from animal to animal. The exact channel of infection is known in a few cases only, and, therefore, each new fact discovered in relation to the transmission of any one is a matter of considerable scientific and economic interest, since its application may be general.

Nature of the disease.—The bacteriologist of the Cape Agricultural Department, Mr Wm. Robertson, in an article which appeared in the

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Agricultural Journal for 5th June 1902, says : " It (malignant jaundice) may be roughly defined as a malarial disease affecting dogs, communicable by direct inoculation from animal to animal, but not by cohabitation or ingestion, and due to the presence of a hæmatozoon in the red blood corpuscles." Dr Hutcheon, the Colonial Veterinary Surgeon, long ago (*Agricultural Journal*, 30th November, 1893) pointed out the similarity of the disease to redwater in cattle and biliary fever in horses ; and we now know that these three diseases are due to three closely related micro-organisms that live at the expense of the red corpuscles, being respectively called piroplasma canis in the case of the dog disease, piroplasma bigeminum in redwater, and piroplasma equi in biliary fever.

The symptoms of malignant jaundice and the *post-mortem* appearances of the viscera of dogs that succumb to it were described at length by Mr Robertson in the article by him above referred to, and I need only mention the superficial characters. The animal attacked first shows illness by its rapidly losing flesh, lacking appetite, becoming listless, and lying quietly curled in one position for hours at a time. If its temperature is being taken regularly, fever will be evident before any other symptom ; but a reading taken when illness is manifest may be normal, as a remission of the fever is characteristic. As the disease progresses, anæmia becomes pronounced, the inside of the eyelids, the gums, and other visible mucous membrane becoming pale and bloodless ; there may, too, be a yellowish tint to these parts, and almost always distinct jaundice shows on the thin skin of the abdomen. If the blood be superficially examined it is seen to be thin, watery, and slow to clot. The urine generally, but not always, becomes blood-coloured. Some dogs die within twenty-four hours of indicating illness by their appearance, but most generally linger on for five days at least, and die from apparent exhaustion after the fever is spent. The most marked features on opening an animal which succumbs are a jaundiced condition of the fatty and connective tissues, a greatly enlarged liver, a still more relatively enlarged spleen, and, generally, reddish urine. Some dogs make a good recovery, and in a month or six weeks are again perfectly healthy ; others remain emaciated for many months, but gradually become restored, whilst still others contract nervous disorders and remain permanently affected.

The dog only is known to be subject to the disease, and no breed appears to be exempt. The better bred the dog, the more severely the disease appears to affect it ; but fatal cases seem to be not uncommon amongst the meanest curs kept by the natives. Young dogs, seemingly, are as severely affected as adults, as in the experiments to be described later on mere pups speedily succumbed to infection. Mr Robertson inoculated a horse, an ox, a sheep, a cat, a fowl, a guinea-pig, a rabbit, a rat, and a mouse with blood from a sick dog, without in any case reproducing the fever. As will later be shown, a jackal was infested with pathogenic ticks and inoculated with virulent blood without its becoming infected.

Occurrence of the disease.—The disease appears to occur throughout the British South African colonies, but to be far less prevalent in high inland districts than near the coast ; this occurrence corresponds with the distribution and numerical abundance of ticks. Dr Hutcheon mentioned that it was known in Europe in his article written ten

years ago, and stated then that it appeared to be more common in the Colony than there. Recently it has received close attention at the Alfort Veterinary School in France, in the southern part of which country and in the warmer parts of Italy it appears to have been well established for many years. Reference to its occurrence in East Africa, and in Senegal on the west coast, is made by Mr Robertson in his paper.

Whether or not the disease is native in South Africa is unknown, but it has certainly been established in parts of the Colony for a long time. Mr Robert Warren, of Avoca, Kei Road, distinctly remembers it as far back as 1844, at Graham's Town. Mr J. B. Currey, of Vergelegen, Mowbray, recollects cases in the Lange Kloof, Humansdorp, in 1853 or 1854. A fatal case at Cradock about 1846, in a pet dog of his, is recalled by Mr W. E. Murray, of Roodebloem, Graaff-Reinet. Several Cape Town people remember that it was common about the city thirty years or more ago, and Dr Hutcheon found it widely prevalent when he came to the Colony in the early eighties. A prominent Natalian, of whom I chanced to enquire, says he has known it ever since he took up his residence in Natal, about 1880. Probably the disease was at the Cape very much earlier than any of these references indicate. In the published letters of Lady Anne Barnard ("South Africa One Hundred Years Ago," p. 110) is a letter from the Cape, dated 29th November 1797, which contains this significant phrase which I italicise: "As we drove along, we saw our only English dog *who has survived the ailment which attacks all who arrive here*, who is a stout vulgar pointer . . . pointing at something." In the lack of any other common and usually fatal disease, there is little reason to suppose the reference not to malignant jaundice.

However, there is a little evidence that the disease has not always been known in districts where it now occurs. Mr H. Dugmore, of Mount Donkin, Bathurst, has told me that when farming near Bowden, in the Albany district, before 1860, his people kept many dogs; and that about the year mentioned the disease appeared, and killed every one. A similar experience was related to me by Mr L. S. de Wet, who has all his life lived near Rawsonville, in the Worcester Division. He said he clearly remembered that many dogs were kept on his father's and surrounding farms in his boyhood; and that about 1860 the disease appeared in the neighbourhood and swept off nearly all of them. Since that time it has been very difficult to rear dogs thereabouts. In both these cases it is possible that the immunity of the dogs prior to the outbreaks might have been due to local absence of the particular tick that carried the disease, or to the non-introduction locally of any sick or salted dog which might start the infection of ticks already present; the local dogs might, perchance, have been brought to the farms as susceptible pups, or been bred from such dogs later.

It appears possible for the disease to be stamped out on a farm. When the fact of its transmission by a tick was published, Mr Robert Warren, above mentioned, wrote:—

"I have been much interested in the account of your experiments with ticks as a means of transmission of distemper amongst dogs. This part of the country has a bad reputation for what we have always termed 'distemper,' so much so that it was difficult to rear

good dogs.   But some years ago a couple of pups introduced red
mange on to the farm, and it was two years before this trouble was
overcome.   Following on the mange, we had a plague of a kind of flea
which would bury itself in the nose and ears of the dogs.   To get rid
of these troubles we resorted to dipping the dogs in Little's sheep dip
and other mixtures.   Now I come to the point that will interest you.
We have to keep a pack of small dogs on account of jackals being so
troublesome.   Several litters of pups have been reared during the last
four or five years, but none of them have had distemper, nor have any
of the grown-up dogs.   Some time ago I remarked that the distemper
germ seemed to be stamped out on the farm, not for one moment
thinking that the germ was conveyed by ticks.   Now I am convinced
that the tick theory of infection is correct, and that the disease was
stamped out by the constant dressing and dipping of the dogs."

It may be that the dipping kept the tick greatly reduced in numbers,
and thus caused an elimination of the infection.   But Mr Warren,
after writing the above letter, made enquiries amongst his neighbours,
and elicited the fact that the disease is not now nearly so serious on
their farms as it was some years ago.   Fifteen years ago, he writes,
only about one dog in five could be reared in the neighbourhood.

The virulence of the infection undoubtedly varies greatly, and even
a mild attack appears to confer a high degree of resistance to a later
infection of a strain that would prove quickly fatal to fully susceptible
animals.   On some farms nearly every dog recovers ; on others nearly
every one, or even every one, dies, many of them after having shown
symptoms of illness for only two or three days.   The variability of the
virulence is probably the true explanation of why remedies which are
apparently infallible with some parties are wholly devoid of value in
the hands of others.   Nearly every farmer and other dog-owner has
his favourite method of treatment.   Some dose with Carter's liver
pills, others with Beecham's pills, castor oil or other purgative, others
with emetics of various sorts.   Haarlemensis or Haarlem oil is a
favourite remedy with many, and not a few extol calomel as the only
one of numerous drugs tested that has ever proved of any service.
The removal of the small tendon under the tongue is a cruel practice
which is widely believed to be most efficacious ; it may perhaps assist
by preventing the poor victim from taking food when food aggravates
the disease.   The Colonial Veterinary Department has experimented
with many drugs without finding any one of much value.   Calomel
seemed to help more than anything else.   Mr R. Bromley, of Ronde-
bosch, says he is very successful with calomel, but he uses it in
tremendous doses.   His plan is to give a pill containing twelve grains
of calomel and twenty of jalap when the disease is first observed ; then,
if no improvement is noticed within two days, a second pill of the same
sort is given, followed a few days later by a third if the case is obstinate.
The strength of pill mentioned is for dogs as large as a year-old pointer.
He gives eight or ten grains only of calomel to small dogs, and may
omit the jalap after the initial dose.   But, whatever the medicines used,
the consensus of opinion is that the sick dogs must be kept warm and
quiet and not harassed with food.   When they begin to mend they
should be fed only on easily digestible food, as warm milk, and that in
small quantities ; and they should not be violently exercised.   I
believe a number of the dogs used in our experiments died, after the

fever was spent, from nothing more than being allowed to eat too freely of the ordinary dog fare.

The Transmitting Tick.—The experiments to be recorded have demonstrated that malignant jaundice is transmitted by the common dog tick of South Africa. This is a species of hæmaphysalis which Professor G. Neumann has pronounced to be H. leachi Audouin. In Professor Neumann's monograph on ticks, this species is recorded from various places in the north, east, south, and west of Africa, and from a few places in Sumatra and Eastern Australia. From his references, the species appears to be almost if not wholly confined to carnivorous animals, the lion, tiger, leopard, wolf, dog, cat, etc., all being attacked by it. Personally, I have only taken it from the domestic dog and cat, but have fed many adults on a jackal. Professor R. Koch, in his first report on African coast fever (see *Cape Agricultural Journal*, July 1903, p. 37), mentions that he now and then came across specimens on cattle at Umtali, Rhodesia ; and Professor Neumann, in his work, states that he received one specimen taken from a horse in New South Wales. About Cape Town it is certainly not common on any domestic animals other than dogs and cats, though now and then single specimens are seen on cattle.

The dog tick is very easily reared. All stages freely attach almost anywhere on the skin of a dog. The larva and nymph both drop from the host to moult. They both feed up quickly, and may drop in less than forty-eight hours from the time they attach ; more unusually, however, they remain from sixty to seventy-five hours. The time consumed in the change from one stage to the next varies with the warmth to which the tick is exposed. In summer the engorged larva may transform to the nymph in ten to twelve days, and the engorged nymph transform to the adult in eighteen to twenty days ; in the winter the same transformations may take ten to fifteen weeks (at Cape Town), but the development will continue and the change take place even if the specimens be kept in an ice-box where the temperature ranges about 55° F. Neither the larva nor the nymph of the species is particularly anxious to hide away in the soil rubbish to moult as is the case with most cattle ticks ; instead, both would rather stow themselves in crevices of wood, bark, or bones, just such places as they would naturally find in a dog kennel or about the lair of a carnivorous animal. Many times I have very successfully moulted them amidst crumpled bits of dry paper within a corked glass specimen tube ; most cattle ticks similarly confined would fail to moult. The two sexes attach without reference to one another, but get together after two or three days. Both sexes move about more or less in quest of more favourable feeding positions, and perhaps also to find mates, as in my experience marked males have moved and settled by females, and marked females moved and settled by males ; but the male appears to be the usual one to move and seek a mate. The females feed to repletion whether or not they are joined by males, but such unmated females have laid only infertile eggs when I have retained them. It generally takes ten days for a female to feed up ; a few specimens are replete by the eighth day, and many hang on from twelve to fifteen. The males remain for several weeks, and may mate with more than one female. The dropped females hide themselves in rubbish, seldom burrowing into

soil as do most cattle ticks, and soon lay their complements of eggs. I have attempted to count the number of eggs in one batch only, and then found about forty-two hundred. At Cape Town, the eggs begin hatching five to seven weeks from the time the female drops in summer, and twelve to sixteen weeks in winter. Three successive generations have been easily reared in fifteen months, but ordinarily probably not over two generations are passed in a year out of doors. No other tick that I have reared is so quick to leave its host at death. There have been few females on any of our dogs at death, but males have been common several times. As soon as the animals began to grow cold, even several hours before death in some cases, the ticks loosened their hold and crawled restlessly amongst the hair, many of them finally reaching some relatively elevated position, such as the point of an ear, and coming to a rest as if there to await a new host ; some crawled quite away from the bodies.

The common dog tick of Europe is generically distinct from the South African species, and, as our tick is not recorded from there at all, it follows that more than one kind of tick must be capable of transmitting malignant jaundice. It may be that more than one kind is concerned in South Africa. Whilst hæmaphysalis leachi is the most generally distributed, as well as the most abundant kind of tick on dogs here, other kinds are not uncommonly seen on the animals in some sections. In Stellenbosch the russet tick (ixodes pilosus) is frequently on dogs, and rhipicephalus capensis is sometimes found. At Nelspruit, in the Transvaal, a few examined were found to carry more adults of the pitted black tick (rhipicephalus simus) than of the ordinary species, and a few adult specimens of the brown tick (rhipicephalus appendiculatus), the bont tick (amblyomma hebræum) and the bont leg tick (hyalomma ægyptium) were found on the same dogs. In tick-rearing work at the experiment kennels, I have found that the blue tick (rhipicephalus decoloratus) may be reared on dogs, and that the early stages of the bont tick and of the red tick (rhipicephalus evertsi) may feed to repletion ; also no trouble at all has been experienced in getting the tampan (onithodoros savignyi) to accept a canine host. Only a very small proportion of the specimens of the blue, red, and bont kinds that have been applied, however, have succeeded in feeding ; and I have thus far wholly failed in attempts to rear larvæ of the pitted black tick on dogs, not one of many thousands applied having fed to repletion.

When and how the Tick becomes Infectious.—The infection of the animal does not originate, of course, from the mere bite of the tick. To communicate the disease the tick must have a virus to transmit that was derived some time before from an animal whose blood was infectious. The tick is normally innocuous except for the slight irritation it causes by its bite and for the trifling quantity of blood which it takes ; indeed hundreds of the tick may and often do attack a susceptible dog and cause no more injury to the health than would so many fleas.

The relation of the disease to redwater in cattle first aroused the suspicion that a tick was the channel through which the disease spread from dog to dog, the fact that redwater was thus spread having been demonstrated by American investigators. Naturally suspicion fixed upon the one common dog tick, and towards the end

of 1900 experiments were started to determine if the suspicion was well founded. The result has been to clearly establish the fact that the tick is the medium of infection. However, the transmission is not so simple as is presumed to be the case in redwater, which is supposed to be transmitted by the larval progeny of ticks off affected animals. The dog tick is found to communicate malignant jaundice only in the adult stage of its life, and then only when in the adult stage of the preceding generation it imbibed infectious blood.

The source of the infection may be either a dog actually ill with the disease or one "salted" to it, that is, a healthy dog immune through having had an attack. Presumably, the recovered dog is a more common source than the actually sick one, since it is about continually. The sick dog dies or recovers in a few weeks; and whilst ill it is apt to remain helpless in its sleeping-place and its vicinity be visited by fowls or birds on the look-out for such dainty morsels as engorged ticks. It is probable, however, that many and perhaps generally most of the progeny of ticks which fall from recovered dogs are not carriers of the virus, and are quite as harmless as the progeny of ticks from susceptible animals. Similarly, it is unlikely that every one of the thousands which arise from a mother of an actually sick dog is pathogenic. Susceptible new dogs often live in localities for several months where all other dogs are salted to the disease and yet escape an attack, although they become much infested with adult ticks that must have come from mothers dropped by the longer resident recovered dogs. Moreover, progeny of ticks off dogs that have lived through the disease have failed to communicate infection in some of the tests which have been made.

A single pathogenic, that is organism-containing, tick is probably ample to communicate infection that may lead to death. The smallest number of ticks actually applied in the tests with the result of producing the disease was ten. This number and twelve were both twice tried with success, and death followed in all four cases. Probably, too, the virulence of the attack induced by ticks depends far more on the quality of the infection which the ticks communicate than upon their numbers. The cases produced by the use of what will be called Lot 4 ticks in the recital of tests to follow were materially milder in type than those resulting from Lots 2 and 3 ticks, although the number of ticks applied was greater.

In determining if the bite of a single tick will transmit the disease, tests resulting negatively would be inconclusive, unless many times repeated. It seems possible that a tick might lose the capacity to infect, or that the infection which it did transmit might fail to become absorbed and reach the blood. Furthermore, it is not unreasonable to suppose that many ticks are quite innocuous despite being the progeny of parents that fed on highly virulent blood. The microscopical examination of virulent blood, that is the blood of an actually sick dog, shows that a very small and variable number of the red corpuscles are invaded by organisms of the disease. The examination of a recovered dog's blood discloses none of the organisms, as a rule ; but, presumably, there are some present, though too few to be observed, as the inoculation of blood from a recovered dog (at least for several months after its sickness) produces the disease in a susceptible dog, like the inoculation of blood from a sick one, with the

difference that a materially larger quantity must be used.   Perhaps, altogether, a third of 1 cc. of blood is sucked up by the mother tick ; and these few drops have to suffice for her own nourishment and for the production of the several thousand eggs which she has to develop. In these vital processes, one to two millions of corpuscles, drawn up whole into the body, may be broken up and used, and if the blood came from a dog at the height of the fever a hundred thousand or more of these corpuscles may have contained disease organisms.   It is likely that a vast number of the organisms perish in the changes, but that those that survive undergo development with multiplication. But, however changed in form and number they become, the organisms must remain very small in bulk compared to the bulk of assimilated food that is ultimately transformed into eggs, and the chances hardly seem good that every one of the thousands of eggs will receive a share in their distribution.   But even if every egg does participate in the division, it is hardly conceivable that enough to secure the infection of an animal will always survive through the periods of embryonic development, the metamorphosis of larva to nymph and nymph to adult, and the often prolonged fasts of larva, nymph, and adult whilst awaiting a host, and then finally will be lodged within the adult whence it will be successfully ejected into the flesh of a dog victim.

Whether or not a tick is injuriously affected by being a carrier of the infection is unknown ; but the individuals of the various lots of pathogenic ticks which have been reared have appeared quite as vigorous, active, and long-lived as the individuals of the lots of non-pathogenic ticks that have been under observation.   There is always a tremendous mortality amongst ticks, probably not one in a hundred hatched living through all three feeding stages, so fine distinctions in the healthiness of different lots cannot easily be drawn.

Cases of the disease have been produced at all seasons of the year through tick infection ; and, so far as could be observed, the virulence was affected neither by the season nor by the duration of the interval between the acquirement of the infection by the tick and the transmission to a new animal.   The metamorphoses of the ticks employed to produce some of the cases were hastened by keeping the specimens at a constant high temperature in an incubator, and those of others retarded by the use of an ice-chest.   Under natural conditions, the disease also occurs at all seasons ; but it is generally much more common during some months than others in any given district, its prevalence doubtless depending on the circumstances influencing the development of the tick.   About Cape Town, the cases brought to my notice have been most numerous in the fall and early winter, that is, during the first half of the rainy season ; but a gentleman residing at Rondebosch, who keeps a number of dogs and who has been familiar with the disease for many years, tells me that more cases occur in October than in any other month.

Young pups have seemed to be fully as susceptible and as severely affected by the disease as adult dogs in the tick experiments and in blood inoculation experiments.   It is a common observation among farmers, however, that dogs are usually several months old before they contract the disease, and that dogs are usually safe from it after they are two or three years old.   The explanation may be that pups

are rarely taken into the veld, and, therefore, are little exposed to tick attack compared with older dogs ; and that dogs two or three years old have generally had the disease. Ticks drop from dogs about the homesteads even more numerously than elsewhere, perhaps ; but there is comparatively little cover for them, and fowls are usually about that will pick them up.

It has been intimated that it is possible that more than one kind of tick can convey the infection. If other kinds do carry it, it' is probably from one generation to the next, and very likely it is then transmitted only by the adult ticks, as in the case of hæmaphysalis leachi. The Bont tick, when fed as an innocuous larva on a heart-water-sick animal, gets infection of that disease, which it will transmit to a susceptible animal when it next feeds ; but the Bont tick, to judge from one test made, is quite incapable of thus deriving and transmitting the infection of malignant jaundice. So also is the Dog tick itself and the Tampan tick. Adult dog ticks taken directly from a sick dog and placed on a healthy dog may or may not give the disease ; but, even if they do, the infection transmitted is probably not from the sick dog from which they were taken, but from the dog on which their parents fed. The direct transmission of infection by a blood-sucking fly, which bites more than one animal within a few hours, is conceivable ; but the indirect and remarkable transmission by the tick is undoubtedly the natural method.

Experiments.

The statements in the paragraphs under the last sub-heading are largely deductions made from the results of experiments conducted during the last three years with the definite object of ascertaining how and by what parasites the disease is transmitted. Their value may be judged by a consideration of these experiments. They are given below, and, to facilitate a ready comprehension of the scope and bearing of each, they are arranged under pertinent headings.

It is proper to explain that the veterinary branch of the department, chiefly through Mr Wm. Robertson, its bacteriologist, has been engaged in studying malignant jaundice from the veterinary and bacteriological standpoints, whilst my office has experimented to elucidate the avenue of transmission. I have pleasure in acknowledging that to the veterinary branch I am indebted for authoritative confirmation as malignant jaundice of nearly all the cases of the disease produced in my experiments, such confirmations being made by examination of blood smears or by *post-mortem* examination of the viscera, or by both. In return for this assistance I have rendered aid to its phases of the investigation in numerous ways, such as providing reared pathogenic ticks for crushing experiments and for sectioning, by assisting at inoculations, and the taking of smears and keeping of temperature and other records of inoculated animals during Mr Robertson's long absences.

The dogs for the experiments were all obtained in or about Cape Town. Many were purchased, but most were procured for the asking from the " dogs' home," the municipal institution to which unlicensed and stray dogs are taken by the police authorities. The municipal storekeeper, Mr Percy Clutterbuck, has my thanks for providing these

animals. Control dogs were always kept, and, as a rule, most of the dogs used were kept in the kennels for a fortnight or more before being brought into an experiment. Dog biscuit, varied with goat flesh occasionally, was used as the food; the young puppies were given diluted condensed milk. Few of the animals were given medicines when ill, the disease being allowed to run a natural course. Sick dogs were generally isolated for their comfort, were kept well supplied with dry beds in the form of bagging, and given warm condensed milk.

A. Infection transmitted by Adult Progeny of the Dog Tick when from a Sick or Recovered Dog.—The first experiment conducted was a test of nymphal specimens of the tick, which as larvæ had been fed on a sick dog. No infection resulting, the progeny of adult ticks taken from presumably infectious sources were naturally tested, the infection of redwater being known to pass to the progeny. The leading facts in regard to the progeny tested, and the dogs on which the tests were made with positive results, were as follows :—

1. Lot 1 ticks, the mixed progeny of females collected 16th December 1900, a few from a sick dog and a few from a long recovered dog. Fed as larvæ during January and as nymphs during March on Dog No. 0.

(*a*) Dog. No. 0. Mongrel pup, born in October 1900. Infested 11th March and on several subsequent days with Lot 1 adults. Fever appeared about 2nd April. Chloroformed when slowly dying 10th April. Diagnosis as malignant jaundice confirmed by *post-mortem* examination and finding of the organism in blood smears.

Lots 2, 3, and 4 were progeny of females removed from actually sick dogs.

2. Lot 2 ticks, the progeny of females taken off Dog No. 0, 10th April 1901, when animal dying. Reared as larvæ and nymphs on Dogs 1 and 2 in June and July.

(*a*) Dog No. 1. Fox-terrier pup, born about 15th May. Infested 27th August with about forty Lot 2 adults. Fever appeared about 8th September, and terminated fatally on the 11th. Diagnosis confirmed by *post-mortem* examination and the finding of the organism in blood smears by Mr Robertson, also by reproducing the fever through inoculated blood in Dogs 3 and 4.

(*b*) Dog No. 2. Sister pup of Dog. 1. Became infested by strays from No. 1, and sickened same day as that animal. Died 10th September. Lesions typical and organism in blood.

(*c*) Dog No. 6. Irish terrier pup, born about 12th August. Infested with five males and five females of Lot 2 adults 23rd September. Fever appeared 5th October, and death followed on the 9th. Lesions typical and organisms in blood. Ten males and ten females of same lot of ticks had been added 25th September.

(*d*) Dog No. 8. Black-and-tan adult mongrel, standing about fourteen inches high at shoulder. Infested with five males and five females of Lot 2 adults 23rd September. Three females and one male at least became attached. Animal isolated in shed by itself, so only the ticks applied could possibly reach it. Fever appeared 4th October, and ended fatally on the 15th. Lesions typical.

3. Lot 3 ticks, the progeny of a female off Dog 6 on 6th October (second day of fever), and two off Dog 8 on 4th October (first day of

fever). Fed as larvæ on Dog 7 in December, and as nymphs on Dog 15 in January.

(*a*) Dog No. 18. Large, heavy, adult fox-terrier. Infested 3rd February with six males and six females Lot 3 adults. Ten males added 7th February. Fever appeared 21st February, and terminated fatally 5th March. Frequent and heavy doses of calomel given, and illness probably thus prolonged. Diagnosis confirmed by *post-mortem* and blood examinations by Dr G. W. Robertson, of Medical Officer's Department.

4. Lot 4 ticks. No female ticks were removed from Dog 18. A fine lot, however, were secured in July 1902 from a pointer dying from fever contracted on the Cape Flats. This dog's case was confirmed by inoculation of blood from it into susceptible dogs. Progeny of the ticks fed as larvæ and nymphs during October and November on Dogs 56 and 57 (young pups).

(*a*) Dog No. 53. Small fox-terrier adult. Infested 9th December with twenty-five male and female adults of Lot 4. About forty more applied 15th December. Fever up 22nd December, and continued until 1st January. Passed reddish urine 2nd to 4th January. Seemed certain of complete recovery on 12th January, and on that date destroyed.

(*b*) Dog No. 56. Small mongrel puppy, born about the middle of September, on which, with No. 57, all the Lot 4 ticks were fed as larvæ and nymphs. In poor health when infested 22nd December with twelve females of Lot 4. Came into fever 1st January, and died 6th January. Opened by Dr Hutcheon and the typical lesions observed.

(*c*) Dog No. 57. Sister pup to No. 56, and, like it, afflicted with worms. Infested 22nd December with twelve females of Lot 4 and isolated in company with No. 56. Came into fever 1st January, and died 5th January. Opened by Dr Hutcheon, and diagnosis as malignant jaundice confirmed.

(*d*) Dog No. 59. Rough-haired adult fox-terrier. Infested 22nd December with thirty-eight females of Lot 4. Fever appeared 1st January, and ran typical course to the 10th. The chances for a complete recovery seemed very good on the 12th, but on that date animal destroyed.

(*e*) Dog No. 60. Small fox-terrier adult. Infested 22nd December with forty-five males of Lot 4. Fever appeared 4th January, and terminated fatally 11th January. Not opened, but jaundice very pronounced and urine red.

(*f*) Dog No. 61. Small fox-terrier adult. Infested 22nd December with fifty-five males of Lot 4. Came into fever 2nd January. Destroyed 12th January, at which date it looked as if almost certain of recovering.

(*g*) Dog No. 62. Small mongrel adult. Infested 22nd December with sixty males of Lot 4. Came into fever 2nd January. Destroyed 12th January. Was not very ill and was vicious to the end.

(*h*) Dog No. 63. Medium-sized fox-terrier adult. Infested 22nd December with ten females of Lot 4. Two of the ticks seen on the skin 27th December, and more probably attached, but *no fever produced.* Infested 7th January with fifteen more females of the same

Lot. Fever appeared 18th January, and ran a typical course of high temperature without much affecting the dog's appetite or checking its bark. Destroyed 12th February.

(*i*) Dog. No. 66. Adult mongrel, the size of a fox-terrier. Inoculated by Mr Robertson with serum 16th January, and on 21st January about forty Lot 4 adult ticks applied to determine if serum gave protection. Fever came up on 1st February, and death took place on the 8th.

(*j*) Dog No. 67. Adult mongrel. Treated in all respects like 66, and fever appeared on same day. Died 14th February. *Post-mortem* examination by Mr Robertson revealed the typical lesions.

(*k*) Dog No. 71. Large Irish terrier adult. Inoculated by Mr Robertson 9th February with blood pressed from ticks taken off sick dogs, but no reaction produced. To test its susceptibility, infested 3rd March with thirty males and females of Lot 4. Came into fever 16th March, and died with typical lesions 22nd March.

(*l*) Dog No. 74. Fox-terrier adult. Inoculated by Mr Robertson 10th February with crushed remains of about fifty Lot 4 adults in water medium. No reaction to 3rd March. Then infested with thirty other specimens of same kind. Fever appeared 13th March and ran the typical course without causing great prostration. Destroyed 31st March, when daily growing stronger.

The following lot was the progeny of females taken off a dog that had fully recovered its health :—

5. Lot 5 ticks. This lot was entirely the progeny of females taken off a salted dog. The animal (No. 54) had had a relatively mild course of fever following blood inoculation, 24th October 1902, and was in normal health and temperature when the parent ticks were fed in early January. The larvæ and nymphs were reared on two healthy puppies during February and March.

(*a*) Dog No. 72. Adult mongrel, size of a large fox terrier. Infested 29th April with about seventy-five adults of Lot 5. Fever appeared 9th May, and animal died on 13th. Blood smears examined by Mr Robertson, and typical organisms found in abundance.

(*b*) Dog No. 75. Adult mongrel. Infested 29th April with about seventy-five Lot 5 adults. Fever came up 10th May, and death followed on the 20th. Fever curve and symptoms typical.

(*c*) Dog No. 76. Adult mongrel. Infested as were Nos. 72 and 75. Fever came up 9th May, and animal died 19th May. Course of fever and symptoms typical.

Some of the surplus ticks of Lot 5 were sent to Dr A. Theiler, the Veterinary Bacteriologist of the Transvaal, who soon afterwards wrote: " I placed the dog ticks on two dogs, and both sickened after about ten days and died of malignant jaundice."

B. *Failures to Infect by Adult Progeny of Ticks from Sick and Recovered Animals.*—Amongst the many dogs to which Lot 4 adult ticks were applied not one failed to become affected, although the strain of the disease transmitted appeared to be much milder than what was obtained from the other lots of ticks. One failure was experienced in the use of Lot 3, the parents of which were taken off sick dogs very soon after the fever came up.

(*a*) Dog No 21. Rough-haired adult fox-terrier. Infested 17th February 1902, with five males and two females of Lot 3. Not

thoroughly examined until sixth day, and then only one tick, a female, discovered. No fever.

(*b*) Dog No 18 that died from the attack of ticks of this lot may not have been infested by the first ticks placed on it ; if it was, the incubation period, eighteen days, was unusually long.

Another failure occurred with the use of a few ticks that were the progeny of a female, taken off a sick dog in the country, which was sent to the Veterinary Department. There seemed no doubt that the host dog was suffering from the disease. The rearing of the progeny was very unsuccessful, only four living to become adult.

Dog No. 7. Small fox-terrier pup. Infested 10th November 1901 with three males and one female. The males attached, but the female was never located. No fever.

These two failures are not worth much consideration, but the following ones, all from one lot of ticks, are very instructive, in showing that a large proportion of the progeny of ticks from infectious sources may chance to be non-pathogenic.

Lot 6 ticks. Progeny of seven females taken off Dog No. 17, 7th to 9th February 1902. Dog 17 was animal infected by inoculation of blood from a recovered dog 7th January 1902. It had fever from 13th January to 24th, on 1st February, 12th to 18th February, 25th to 27th February, and 12th to 14th March. It went "off feed" 14th January, and took almost no food, except milk, until 1st March, after which it took a little biscuit daily. All along after the first onslaught of fever it was extremely anæmic, and no hopes of its recovery were entertained until March. On the 12th and 13th of this month it was tempted to eat a little cooked meat, but fever came on at once and death occurred on the 16th. Organisms of the disease were found by Dr G. W. Robertson, of the Medical Officer's Department, in smears taken on 14th January, and on many subsequent days to and including 6th February, and some also were observed in smears taken 17th February. Therefore organisms were certainly in the blood in observable numbers whilst the parents of Lot 6 ticks were feeding. Lot 6 larvæ and nymphs fed on three puppies in March and April respectively.

(*a*) Dog No. 36. Small poodle. Infested 4th June with thirty-five adults of Lot 6 ticks. No fever. Animal destroyed 1st August.

(*b*) Dog No. 37. Black mongrel, size of bull-terrier. Infested 4th June with about forty adults of Lot 6 ticks. No fever. Proved susceptible four months later by inoculation with 1 cc. of blood from a recovered animal.

(*c*) Dog No. 45. Adult pointer. Infested 4th June with forty males of Lot 6 ticks. No fever. Proved susceptible by inoculation in July with blood from a recovered animal.

(*d*) Dog No. 46. Small yellow cur, adult. Infested 5th June with thirty males of Lot 6 ticks. No fever. Later proved susceptible by inoculation with blood from a sick dog.

(*e*) Dog No. 47. Adult bull-terrier. Infested 18th June with thirty adults of Lot 6 ticks. No fever. Proved susceptible in August by inoculation from the pointer off which the parents of Lot 4 ticks were taken.

A number of engorged nymphs of Lot 6 were sent to Dr G. H. F. Nuttall, of Cambridge University, England, who kindly undertook to

test the resulting adult ticks on dogs confined at his laboratory. As at the Cape, no case of fever resulted, which was greatly to my disappointment, since the object I had in view was to secure over-sea affirmation by a well known scientist that the disease was tick-transmitted. Before he reported his failures I had written as follows: " If failure results we will have evidence that not all the individual progeny of females fed on pathogenic blood are infective. This is what reasoning would lead one to expect, and what I believe is indicated by the results of inoculations with recovered blood. . . . Now the female tick takes up only a small amount of blood, and is it not reasonable to suppose that the infective matter therein is insufficient for the whole of the several thousand eggs which she has to lay ? Perhaps only a very small proportion receive the organism, and it might easily happen that no infective ones be present in the twenty or thirty of the progeny we are finally able to test. In all my previous tests of pathogenic ticks I have had progeny of females taken off dogs during the fever period, when, as we know from microscopic examination of the blood, the organisms are very abundant. This last time the females were removed after a fever period. The blood of the animal at the time was very thin, and probably contained the organisms in small numbers only."

One failure was later experienced with Lot 4 ticks on Dog 63, as recorded under 4 (*h*) above. Two female ticks certainly attached, and the dog sickened later when infested with more ticks of the same lot.

Still another failure was experienced when working with Lot 5 ticks, which traced to a recovered dog.

Dog No. 65. Small fox-terrier, a young adult. Infested 29th April 1903 with about seventy-five adults of Lot 5 ticks. Infestation repeated 5th May, but still not over eight ticks observed on the skin, only two of which, both males, known to date from first application. No fever to 23rd May, when animal destroyed because much affected with mange. Though not proved susceptible it probably was so, as it was town-bred, and had been brought to the kennels, when still a pup, in January. It was a short, hard-haired animal, and the ticks applied which failed to attach probably wandered off to Dogs 72, 75, and 76, which were confined in the same room and were under the same test; all three had skins better adapted to the comfort and safety of ticks.

C. Adult Progeny of Dog Tick fed during Incubation Period of Disease Non-pathogenic.—The tests in this direction were few but decisive. The ticks used were all the progeny of females taken off Dog 6 on 2nd and 3rd October, two to three days before the said dog came into fever from the application of these very ticks, with a few others of the same history. However, as the progeny were fed on various dogs as larvæ and nymphs, they must be classed in several lots for discussion.

Lot 7 ticks, the progeny of females off Dog 6, 2nd and 3rd October. Fed as larvæ on Dog 11, six weeks recovered, about 1st December 1901, and as nymphs on Dog 7, susceptible, about 3rd January.

Dog No. 15. Young fox-terrier. Infested 4th February with eighteen Lot 7 adults. Biting very good, and five of the females recovered after engorgement. No fever.

Lot 8 ticks, same parentage as 7, but fed as larvæ on Dog 12 about 18th December, when animal in full fever, and as nymphs on Dog 14, susceptible, 2nd to 4th January.

Dog No. 15. Infested 17th February with twelve adults of Lot 8 ticks. Biting very good. No fever. Dog later proved susceptible by inoculation of 1 cc. blood from No. 11, injected 17th April.

Lot 9 ticks, same parentage as 7 and 8, but fed as larvæ and nymphs on Dog 7 in health.

Dog No. 16. Small mongrel pup. Infested 27th January with fifty adults of Lot 9 ticks. Biting good. No fever. Died in March as result of heavy inoculation.

D. Nymphal and Adult Dog Ticks fed in previous Stage on Sick or Recovered Dogs Non-pathogenic.—It will be observed that Lot 7 ticks, though fed as larvæ on a recently recovered dog, failed to infect the susceptible dogs upon which they were fed as nymphs and adults ; also that Lot 8 ticks, though fed as larvæ on a dog in full fever, failed to become pathogenic. These results must signify that the larva is as incapable of deriving infection that it can pass on for transmission by a later stage, as it is of transmitting infection that is in it by inheritance from the parent. Further evidence to the same effect, and also that the nymph is similarly innocuous, is afforded by a few other tests that were made.

Lot 10 ticks. The progeny of two females taken off Dog 7, 2nd November 1901, when animal healthy and susceptible to malignant jaundice. Larvæ fed on Dog 12 in fever and dying, 16th to 19th December.

Dog No. 14. Adult fox - terrier bitch. Infested heavily 8th January with Lot 10 nymphs. Eight recovered. No fever. Animal proved susceptible by inoculation from recovered Dog 11 one month later ; it fevered and died.

Dog No. 16. Small mongrel pup. Infested 16th January with Lot 10 nymphs. Eight recovered engorged up to the 20th, when animal washed to destroy balance, probably a score or more. No fever.

Lot 11 ticks. The progeny of Lot 9, and therefore positively non-pathogenic by inheritance. Parents off Dog 16, 5th to 7th February 1902. Larvæ fed on healthy pup, 17th to 20th March. Nymphs fed on Dog 25, 9th to 14th April, just after fever had lapsed in the animal and a few days before its death.

Dog No 43. Adult fox-terrier. Infested 4th June with seventy-five Lot 11 adults. About ten only found to have attached. No fever resulted. Died 15th July from worms and debility.

Dog No. 44. Adult fox-terrier. Infested 4th June along with No. 43, and about same number found to attach. No fever. Animal killed by another dog on 29th June.

E. *Adult Dog Ticks removed from Dogs dying of the Fever may or may not be Pathogenic.*—The tests here grouped prove very well that the tick is not a mere mechanical transmitter of the disease organism ; and one is, therefore, led to presume that it is the true host of the organism of canine malaria.

1. Dog. No. 7. Fox-terrier pup. Infested 9th October 1901 with ten males taken off Dog 6 at its death on the same day. Five at least bit in and remained a fortnight. No fever. Animal later proved sus-ceptible by inoculation of blood from recovered dog.

2. Dog No. 37. Fox-terrier adult. Infested 26th July 1902 with about fifty males taken off dying pointer (source of Lot 4 ticks). Biting good. No fever. Susceptibility proved by reaction to inoculation of 1 cc. blood from Dog 11 recovered.

3. Dog No. 42. Small fox-terrier adult. Infested 26th July 1902 with about fifty males taken off dying pointer. Biting good. No fever. Later proved susceptible by inoculation of virulent blood.

4. Dog No. 46. Small yellow cur pup. Infested 25th July 1902 with about fifty males taken off dying pointer. Biting good. No fever. Later proved susceptible by inoculation of virulent blood.

5. Dog No. 70. Small fox-terrier adult. Infested 9th February 1903 with twenty males of Lot 4 ticks off 66 when dying. Infested 14th February with thirty-five more of same lot but off 67 dying. A few known to have bitten and remained. Fever appeared 24th February; death with typical appearances 4th March.

The source of the infection transmitted to Dog 70 is presumed to have been not the feeding on Dogs 66 or 67, but the feeding of the mother ticks on the pointer. In other words, it is supposed that some one or more of the ticks applied had not exhausted the supply of inherited infection in infecting 66 or 67 ; and that, incidental to attaching to the new dog, some of this old store was injected.

F. *Jackal naturally Immune to the Disease.*—A natural desire of long standing to test the susceptibility of a jackal to the disease was gratified in January of this year, through the kindness of Mr Robertson in placing at my service a young red jackal that had come into his possession.

Jackal. Infested 30th January 1903 with about two hundred adults of Lot 4. Biting very good, and nearly a hundred engorged females secured eight to fourteen days later. Temperature taken daily but no fever readings recorded.

Not being certain that the animal had not had the disease early in life, I had Mr Robertson inoculate it with virulent blood on 21st February, and on 4th March inoculate 8 cc. from it into a susceptible dog, No. 76. The dog did not react, and, as when it was later exposed to pathogenic ticks, it sickened and died, it is clear that the jackal was quite immune.

G. *Tests with other kinds of Ticks, and Fleas.*—The following tests supplied further evidence that the transmission of the disease by ticks is not a mere mechanical transference of the causal organism from one dog to another, as in inoculation with blood.

Bont tick (amblyomma hebræum). Larvæ fed on Dog 14 13th to 18th February 1902, when animal in high fever. As nymphs about one hundred and fifty placed on Dog 21. Biting very good, but all except a few scratched off before engorgement. No fever reaction.

Tampan tick (onithodoros savignyi). Nymphs and adults fed 14th November 1901 on Dog 10 when it was dying in fever. After moulting, five fed on Dog 22, 17th February. No fever reaction.

Another lot fed 18th February 1902 on Dog 14 when it was in high fever. After moulting, about fifteen fed on Dog 42, 1st July. No fever reaction. More of same lot applied to Dog 46, 18th September. No fever reaction.

Fleas (pulex serraticeps). Dog infested 17th December 1901, with five females and three males taken from Dog 12 in fever, and seven-

teen females and six males from Dog 11 recovered. The fleas bit in very well, but no fever resulted.

Reared adult fleas from eggs dropped by females on Dog 9 at height of fever, 1st December 1901, and Dog 11 recovered on same date. Exposed Dog 16, a young pup, to attack by these reared fleas, twelve tracing to Dog 9, and thirty to Dog 11, on 4th January. Fleas bit well. Both this Dog and No. 7 had been thoroughly washed and isolated a few days before the fleas were applied and were seen to be clean of other fleas when the tests were started. Dog 16 transferred 11th January to a room that had previously been occupied for several months solely by recovered Dog 11, and which was swarming with fleas. No. 16 became very badly infested and remained so until washed again on 20th January, but it did not contract the disease.

The transference of lice from sick dogs to healthy ones was not attempted, but on three occasions when very lousy dogs were used in inoculation experiments, clean susceptible dogs were confined in the stalls with them after the fever appeared. The new dogs became much infested with lice without contracting the disease.

During the coming year, if more important work does not interfere, it is proposed to determine if the progeny of hæmaphysalis leachi ticks off sick dogs will prove pathogenic when adults, if they are fed on a cat when larva or nymph. And it is further planned to test adult progeny of ixodes pilosus ticks off sick dogs.

PRIMARY ADENO-CARCINOMA OF THE LIVER.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

THE writer has found the condition which is here described under the term of primary adeno-carcinoma of the liver most frequently in Irish cows. The affected animals were consigned from Inverness-shire and Argyleshire, and from the north and north-west of Ireland.

During the year 1903 there were slaughtered in Moore Street Abattoir, Glasgow, 39,704 cattle (bulls, oxen, cows, and heifers), and of these 119 were found to be affected with primary adeno-carcinoma of the liver. Of the total found affected, 116 were cows consigned from Ireland, whilst the remainder were those of animals which, if not bred, had at least been fed for some time, in Scotland.

A few cases have also been observed in sheep. Enquiry failed in all but one instance to elicit where these animals had been reared and fed. The sheep in question was stated to have been consigned to the Glasgow market from West Perthshire. Some of these animals had the appearance of being in perfect health, whilst others were more or less emaciated.

Primary adeno-carcinoma of the liver is seen more frequently during the middle and latter periods of existence. It is frequently associated with adenoma, chronic venous congestion, cirrhosis, distomatosis, cavernous angioma, or, but most rarely, tuberculosis.

As a rule, only one primary neoplasm is present and it may be found situated in any part of the liver. These neoplasms frequently attain large dimensions and cause enlargement and distortion of the

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organ [1].   When the neoplasm is superficially situated, its periphery
rises above the contour of the liver either at an acute angle or as a
gentle upheaval.   Its surface is, in a greater or less degree, undulating.
That portion of Glisson's capsule covering it is either uniformly
thickened and opaque, or the increase in density is confined to the
hollows, whilst that covering the apex of the undulations is so thin
and transparent as to reveal the colour of the underlying tissue.
Portions of liver parenchyma may be present in the saucer depressions
formed by the unevenness of the surface.   These areas of liver tissue
have become isolated through the unequal growth of the neoplasm.
Numerous blood vessels are present in the fibrous capsule.   The
majority of these vessels appear larger than they really are owing to
their being compressed through the tension created by the growth of
the neoplasm.   These tumours, on transverse section, are seen to be
either round, oval, or irregular in shape ; the latter being due to the
presence of buds in the vicinity of the primary neoplasm, or of thrombi

FIG. 1.

The hepatic vein opened, showing a number of small thrombi protruding through the orifice of
as many branches ; these have originated from a greyish tumour lying underneath.

in the lumen of the blood vessels.   Their margin is always sharply
defined, and it is frequently wavy or irregularly crenated.   A more or
less distinct fibrous layer may or may not be found interposed between
the tissues of the neoplasm and those of the liver.   This layer may
encircle the neoplasm and attain an equal thickness throughout, or it
may be unequally distributed, being in places extremely delicate or
even absent.   It, when well marked, may be observed to be formed in
layers lying parallel to the margin of the neoplasm, and between its
different strata are present blood vessels and isolated areas of atrophied
liver tissue.   These neoplasms are divided into lobes by more or less
distinct fibrous septa.   The lobes, which vary in size from a pea to a
cocoanut, tend to be round in shape.   The cut surface of the lobular
tissue becomes slightly rotund, and it has a smooth, glistening, homo-
genous, and somewhat translucent appearance.   It may either be
greyish-white, yellow, green, or brown, and it is not uncommon to see
the tissues of a neoplasm, indeed those of a lobe, show different

[1] A liver thus affected was found to weigh over 80 lbs.

shades of one or more of these colours. The variety and richness of colour of these tumours render them unique among pathological conditions. The consistency varies from the equivalent of brain to that of liver. Variations in the degree of density are found to exist not only in individual neoplasms, but also between the different lobes of the same tumour. The lobular tissues of some neoplasms are seen

FIG. 2.

Section through a greyish-coloured neoplasm, showing the liver tissue at the margin pressed aside, and the wall of the blood vessel at one part thinner than normal, with the neoplastic tissue protruding into its lumen as a rounded elevation. Note also the lobulated appearance of the cut surface.

to be intersected by extremely delicate lines which ramify in all directions. Numerous blood vessels are visible in the lobular tissue. There lumen, on section, remains patent, and from the orifices, the shape of which depends on the angle at which the vessel is severed, blood oozes and masks the colour of the tissues of the tumour over which it flows. They do not possess, as a rule, a visible wall, and consequently they appear to be simply channels formed in and by the lobular tissue. Hæmorrhages are of frequent occurrence. What

appear to the unaided eye to be greatly dilated blood vessels or spaces are found frequently grouped together, imparting to the area an appearance similar to that of a cavernous angioma, grossly

FIG. 3.

Posterior surface of liver of a cow, showing thrombus in portal vein. The neoplasm, which was greenish in colour, occupies the left lobe of the liver.

exaggerated; the fibrous septa of the latter being represented by bands of the lobular tissue. These spaces occasionally contain, instead of blood, a clear watery fluid more or less tinged with red. The tissues in the vicinity of these hæmorrhages are frequently stained by the dissolved colouring matter of the extravasated blood.

The neoplastic tissues tend to invade the liver substance either as buds or as secondary lesions, or to gain the lumen of the blood vessels and form thrombi, or to extend to the lymphatic glands.

The liver tissue in the vicinity of the tumour may be invaded, and from the arrested migrant neoplastic tissue there originates a lobe. This process of budding indicates the mode by which the tumour extends, and, also the origin of the lobulation of the main mass. As the new lobe increases in size the adjacent liver tissue is pressed back on all sides, undergoes atrophy, and is replaced by fibrous tissue. The intervening hepatic tissue between the neoplasm and its new lobe becomes ultimately wholly destroyed and replaced by fibrous tissue,

FIG. 4.

Section of the lung of a cow showing numerous lesions, varying in size from a pea to a millet seed, and secondary to a neoplasm in the liver. In the lumen of pulmonary vein is a thrombus.

which forms a septum dividing the tissues of the new lobe from those of the pre-existing ones. This process of budding may be repeated indefinitely, and the neoplasm (which originally could not be distinguished from an adenoma) may attain to a large size.

Smaller tumours may be present throughout the substance of the liver possessing macroscopic characters similar to those of the larger or primary neoplasm. Some of these smaller growths (adenomata) may have originated independently of the larger, whilst others are undoubtedly the result of the infarction, or of the migration of neoplastic cells. On several occasions, on dissection, an embolus was found fixed at a bifurcation or in the lumen of one of the smaller branches of the portal vein. The embolus was evidently the detached

portion of a thrombus situated in closer proximity to the portal fissure, which had become detached and carried by the blood stream to its point of arrestment. Careful examination demonstrated that the tissues of the embolus had not only become adherent to the inner coat of the vessel, but had also invaded the liver parenchyma adjacent to the attachment.

The manner in which the tissues of these neoplasms burst through the limiting fibrous membrane is well seen when the walls of adjacent blood vessels become involved. The neoplastic tissues in places gradually increase in bulk, press upon the wall of the vessel, and ultimately cause a more or less rounded upheaval to project into its its lumen. The wall of the vessel superposed to these upheavals gradually becomes thinner and eventually ruptures. In other cases
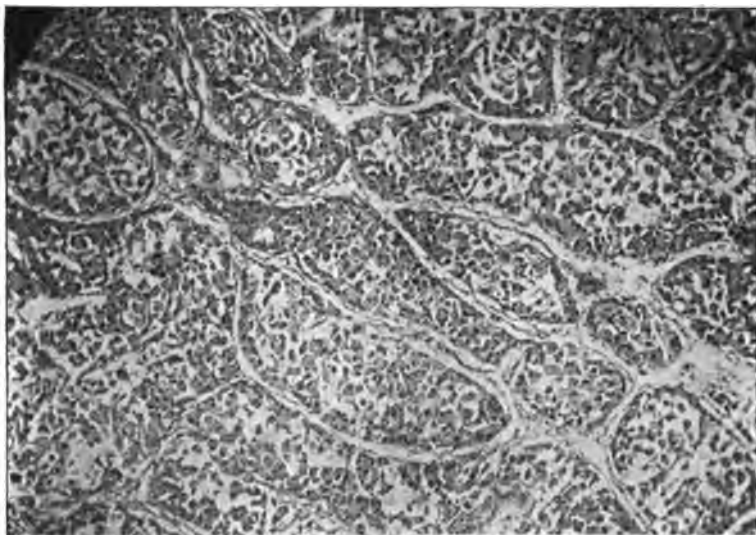
FIG. 5.

Section of pale-coloured neoplasm, showing large alveoli with scanty stroma and delicate-walled blood vessels (×100, van Gieson stain).

the cells of these neoplasms gain access to a small tributary or branch, multiply and finally emerge into the lumen of the larger vessel as a minute rounded body. These thrombi at first project into the lumen at a right angle to the wall of the vessel, but as they attain larger dimensions become flattened and fan-shaped. Some of these thrombi grow to a large size, and it is not uncommon to find one with a diameter of 5 centimetres. Each thrombus can be divided into processes which are interwoven and matted together. Their structural arrangement is more easily observed in those of small size ; these are seen to be foliated, each folium having a free rounded extremity and a fixed base. The folia or processes which go to form the larger thrombi cannot, however, in every case be differentiated, as a tension created by the growth of the thrombus in the lumen of the vessel moulds their soft plastic tissues into what appears to be a solid mass, but which can be split up into its component parts by dehydration in alcohol. The

thrombus conforms to the ramifications of the containing vessel, and an offshoot from the former may be seen to penetrate the lumen of even the smallest branches of the latter. They do not, as a rule, form attachments to the walls of the vessels but instances are to be met with in which more or less intimate adhesions have occurred. Their surface, which is smooth and glistening, appears to be covered by an extremely delicate transparent limiting membrane, but this on mani-
. pulation with forceps is found to be non-existent, the eye having been deceived by the appearance of the tissues. The tissues of the thrombus possess the same macroscopic characters as those of the neoplasm. Through the tension created by the growth of the tissues of the thrombus the calibre of the containing vessel is increased, its walls are dilated, and the adjacent liver parenchyma is atrophied. It is interesting to note that thrombi are more frequently found lying in the lumen of the branches of the portal vein than in those of the hepatic artery or vein.

A portion of the thrombus is liable to become detached and carried by the blood stream to a distance, there to be arrested and form a new focus of disease.

This can be more easily observed in the case of a thrombus formed in the lumen of the hepatic vein, where portions of it becoming detached are carried away by the blood stream, to be arrested in the branches of the pulmonary artery. These emboli retain their vitality, and eventually form secondary lesions in the lungs. These lesions vary in size from a millet seed to a walnut. They are round, and, if superfically situated, project beyond the contour of the collapsed lung. They are intimately attached to the adjacent pulmonary tissues, and are, as a rule, firm in consistency. There margin is seen, on section, to be sharply defined, and is in the majority of cases of a deep red colour. The colouration of the margin passes imperceptibly into that of the tissues of the nodule, which may either be greyish-white, yellow, green, or brown.

A detached particle from a thrombus in the hepatic vein may traverse the pulmonary capillaries, gain the systemic circulation, and produce its effect in the tissues of the organ in which it is arrested.

In one instance a metastatic lesion was found in the cortical substance of a kidney. It was not observable until the kidney had been cut. It was then seen to have attained the dimensions of a hazel nut. Its margin was sharply defined and irregularly crenated. Its tissues were soft in consistency, green in colour, and freely interspersed with blood spaces.

The walls of the bile ducts or gall bladder are liable to become involved when the neoplasm is situated in their vicinity. The tissues of the neoplasm after destroying the walls gain the lumen and there form a thrombus. In a case in which the gall bladder was implicated the tissues of the neoplasms had burst through at the cervix and had formed a villous-like growth which acted as a valve, and, whilst permitting of the ingress of bile, it effectually prevented its egress. This growth, which presented similar macroscopic characters to those of the neoplasm, was irregular in shape and measured 7·5 cm. by 5·0 cm. The gall bladder and its contents weighed 2·15 kilo. In its interior was an inodorous mass, soft and fibrous in consistency, and dirty green in colour. The bile was normal in colour and in consistency.

This condition differs from carcinoma in that metastasis occurs less

frequently by the lymphatic system than by the blood. The lymphatic glands most frequently affected are those of the hepatic group. The affected glands may either be normal in size or much enlarged and distorted.[1] On section the glandular tissue is seen to be displaced, and in extreme cases it remains merely as a more or less thin layer at the periphery. The macroscopic characters of the tissues of these secondary lesions are similar to those of the primary neoplasm.

The tissues of these neoplasms are prone to undergo degenerative changes. These may be manifested by the affected tissue assuming a yellowish tinge. In other cases they are revealed by the presence of necrotic patches which vary in size from mere specks, all but invisible, to areas involving the whole or part of the tissues of a lobe. The margin of the latter is seen, on section, to be irregular, sharply defined, and raised somewhat above the adjacent tissue. A small quantity of a glairy greenish-brown semi-fluid material is frequently found interposed between the tissues of the necrotic area and those of the neoplasm. The mass of dead tissue can, without much difficulty, be enucleated. It is, as a rule, of a yellowish colour, but towards the margin assumes a greenish tint. It is somewhat granular in appearance, similar to newly-made cheese in consistency, and devoid of blood vessels.

It is evident, from the resistance to the passage of a knife and the characteristic grating sound emitted, that a deposition of lime salts occurs in many of these areas. Litten has experimentally shown that the precipitation of lime salts is intimately associated with the coagulation of the albumen of a tissue.

Gangrene is occasionally observed. In the few cases which have come under the writer's observation the tissues forming the central zone of the neoplasm were those involved. The living and dead tissues were sharply differentiated. The latter were greenish-black in colour, granular and disintegrated in appearance, and emitted a peculiar pungent and offensive odour. The microscopical examination of a smear preparation from the sphacelus of one neoplasm revealed the presence of large bacilli which were rounded at the ends, were stained by ordinary aniline colours, and frequently showed a large oval spore situated either at the centre or at the ends.

The tissues of these neoplasms may occasionally be found to have undergone myxomatous degeneration. That part of a neoplasm in which the extravasated blood of numerous small hæmorrhages has been replaced by a clear watery fluid may present to the unaided eye much the same appearance as an area undergoing myxomatous degeneration, necessitating a microscopical examination to determine with certainty its condition.

Occasionally the fibrous septa dividing the neoplasm into lobes are found to have attained a very considerable thickness, and the tumour may then acquire a consistency comparable to tendinous tissue. This increase in the thickness of the septa undoubtedly tends in some measure to prevent the extension of the disease. This opinion is based on the fact that the tissues of the lobes of the involved area are seen to be almost invariably undergoing degenerative changes. This change, which is more frequently seen in tumours situated in the left lobe of the liver, is perhaps the result of some irritant causing an

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[1] On one occasion a hepatic gland was found to measure 15 cm. by 4 cm.

increase in the amount of the fibrous elements of the septa of the tumour, or, and more probably, it is merely an extension of a pre-existent cirrhosis, among the ramifications of which the tissues of the more rapidly growing neoplasm had become insinuated.

Microscopical examination of preparations made from fixed and hardened pieces of these tumours shows that the neoplastic cells bear a resemblance to normal liver cells. They are either round, oval, or angular in shape. The nucleus, which is large and round or oval, may contain one or more nucleoli. The cells are arranged in alveoli which vary greatly in size. These alveoli, even when composed of a single or a double row of cells, show no tendency to a regular acinous arrangement, although in places a number may be seen to converge towards a space, limited by endothelium, comparable to a central vein. The stroma is very delicate, and occasionally may show areas invaded by small round cells. It is observed at the margin of the tumour to be continuous with the fibrous capsule. The blood vessels, which are accommodated in the bands of stroma, are numerous and loosely supported. They vary in size from capillaries to vessels of considerable calibre. Their walls, which in some instances are in contact with the cells of the alveoli, are delicate, and in many cases are formed by a single layer of endothelium. They are, in consequence, extremely liable to rupture. A single cell or a cluster of neoplastic cells is occasionally seen surrounded by blood corpuscles in the lumen of the blood vessels. The presence of these free .wandering cells in the blood stream affords a clue to the mode by which metastasis frequently occurs. The microscopical examination of a portion of a neoplasm described as having a naked-eye resemblance to a cavernous angioma showed the blood spaces to be either dilated blood vessels with delicate walls or more or less recent circumscribed hæmorrhages. These spaces vary in size, and are either round, oval, or irregular in shape. They are, as a rule, sharply differentiated from the surrounding tissue. The endothelial lining of some of these spaces is seen to be intact, whilst in others it is incomplete. The cells in the immediate vicinity undergo atrophy and degeneration. These spaces may be separated by a single layer of cells. In other cases the blood corpuscles, as they escape from the ruptured blood vessels, infiltrate the neoplastic tissues. The extravasated blood tends to undergo coagulation, the cellular elements become disintegrated and absorbed, and ultimately only a clear serum remains. The latter is, in many instances, distinguishable to the unaided eye. Crystals of hæmatoidin are frequently found lying free in the tissues.

The margin of a recently formed lesion may be irregular and indefinite, the neoplastic cells being intermixed with liver cells, and the latter in various stages of degeneration ; but in the more mature lobes it is, as a rule, sharply defined. It appears that the neoplastic tissue in increasing in bulk presses back the liver tissue. The usual hepatic arrangement in the vicinity of the tumour is more or less obliterated, and the liver cells tend to become arranged in rows parallel to the contour of the neoplasm. The intervening capillaries remain patent and prominent. The cells become flattened or deformed and ultimately disappear, whilst the connective tissue framework and blood vessels become proliferous. The gradual transition from the liver elements to fibrous tissue can be observed in the capsule or septa,

older. or more mature fibres being in contact with the neoplastic tissue. The fibrous layer, which may attain a considerable thickness, retains a laminated structure, and undoubtedly when it is well marked tends to check the further advance of the disease. In this fibrous layer are present numerous blood vessels. From those interposed between the capsule and the tumour branches are given off to form the vessels ramifying in, and supported by, the stroma. Occasionally bile ducts lined by slightly columnar or cubical cells are to be seen in the capsule or in the septa. • In places the neoplastic cells may be found penetrating as finger-like processes between the lamina of the limiting fibrous tissue.

These neoplasms extend by the formation of new lobes in the liver tissue in the vicinity of the margin of the tumour. These new lobes are a replica of the older lobes. The distance intervening between the recently formed lobes and the neoplasm proper varies in each case. The thickness of the limiting fibrous tissue, which forms a segment of the capsule or a septum according to position, is thus dependent not only on the size attained by the lobe but also on the amount of intervening liver tissue. The latter, as has already been stated, undergoes, as the result of pressure, atrophy accompanied by proliferation of the fibrous elements.

The nucleus of these neoplastic cells are frequently found to have undergone chromatin degeneration. This form of degeneration is unequally distributed; only one cell may be attacked, or a number of cells in an area may be involved. It is manifested by the nucleus staining deeply with nuclear stains. In some instances the nucleus in the presence of a stain of which hæmatoxylin is the active principle is almost uniformly of a deep blue-black ; in others, whilst the outline of the nucleus is still retained, the nuclein is gathered in centres ; or, again, in more advanced cases all evidence of structure may have disappeared, and the centres or masses of nuclein dispersed, but, more frequently, they have coalesced to form a granular mass. These granular masses are frequently the only indication of the previous existence of cells which had undergone degeneration. They may when examined under a low power be mistaken for round cells, but with a higher magnification their granular structure can be determined.

Fatty degeneration of the cells is of frequent occurrence. This change is probably the result of malnutrition resulting from interference with the blood supply through the presence of a thrombus in the vessel of supply. These thrombi frequently attain such dimensions as to all but occlude the lumen.

The stroma is frequently found to have undergone myxomatous degeneration. This degeneration would appear to have a preference for the adventitious coat of the small arteries, the latter being frequently seen isolated in a transparent hyaline basis. The neoplastic cells may through extension from the stroma become involved and a myxomatous space be formed.

The whole or part of the tissues of a lobe may undergo coagulative necrosis. The margin of the affected area is sharply defined. The involved area is very granular. The cell substance is glassy and homogeneous in appearance. The nuclei appear as speckled bodies. The specks, which vary in size, are more or less indistinct, some,

indeed, being all but invisible. Ultimately all structure is entirely obliterated.

The microscopical structure of the thrombus and of the secondary lesions in the liver or lungs is similar to that of the primary neoplasm, and their tissues are liable to undergo the same retrograde changes. The manner of growth is well exemplified in the case of the pulmonary lesions, where the lung tissue can be observed to have been pushed aside to accommodate the new formation, the displaced tissue, as in the liver, forming the fibrous capsule.

HORSE-SICKNESS EXPERIMENTS.[1]

By Dr. A. THEILER, Government Bacteriologist, Pretoria, Transvaal.

THE blood of a horse suffering from horse-sickness is virulent. The injection of virus into susceptible horses produces either dikkop or dunkop. Injection into the jugular vein causes the disease to break out after a shorter incubation period than by injection under the skin.

Minimum quantity of virus varies somewhat for different horses, and ·01 cc. does not under all circumstances produce the disease. One cc. was a fatal dose in all experiments. The injection of virus into the trachea is not followed in every instance by the disease. The application of virus by the mouth produces the disease, but not in every instance, and only when the quantity is 200 cc. or above. The virus when kept liquid retains its virulency for several years.

Virus does not seem to suffer from putrefactive microbes. Virus is dead as soon as it is dry. Virus dried at the normal temperature of the room is destroyed.

Animals which recover from horse-sickness are immune. This immunity is not absolute for every horse. The immunity may break down. Relapses do not as a rule lead to death. The experimental investigations showed that such relapses occurred in 33·3 per cent. of cases after exposure in the worst locality during a period of four months. The mortality from relapses was 11·1 per cent. under the same conditions.

The serum of an animal which has recovered from the disease has no preventive value whatever. The serum of an immune animal which has periodically been hyperimmunised acquires immunising properties.

Immune serum mixed with virus (1 cc. taken as normal lethal dose) and injected subcutaneously does not produce the disease, while the same mixture injected into the jugular vein gives the disease.

Virus which has been in contact with serum for four days, when separated from the serum by a centrifugal machine, will still produce the disease when injected under the skin. The injection of serum causes a passive immunity which may last up to four weeks, but which is usually over after a fortnight. Serum injected before the virus will prevent the disease when the injection of virus is made twenty-four hours later under the skin, but does not in every case prevent the disease when the injection is made into the jugular vein.

[1] Communicated to the Transvaal Veterinary Association at Pretoria on 19th March 1904.

Simultaneous injection of serum and virus, both subcutaneously, will prevent the disease completely when virus is used in small quantities (1 cc.), but not when large quantities (about 50 cc.) are used. But even with the larger quantities the animal will recover in many instances. Simultaneous injection of serum and virus, the virus injected intrajugularly, does not prevent the development of the disease, but the animals will recover in the majority of cases. Injection of serum some hours (three to six) after the virus has been injected does not prevent the development of the disease, but the animal will in the majority of cases recover. Animals which have passed through a reaction after an injection of virus and serum acquire active immunity. An injection of 5 cc. virus after fifty-six days has shown that this immunity is as active as the immunity acquired in the natural way.

The disease produced by the inoculation of virus and serum in any of the ways already indicated is in every respect identical with the disease which arises spontaneously and from which the animal recovers.

Symptoms of dikkop occur in about 50 per cent. of all cases. They appear with the fall of temperature.

The duration of the disease caused by inoculation lasts about twelve days. The length of the incubation and sickness lasts about three weeks. The injection of virus in the form of blood causes hæmolytic properties of the serum of some of the animals injected. Serum of such animals injected into healthy animals produces dissolution of blood and hæmoglobinuria.

The rise of hæmoglobinuria is not alone due to a hæmolytic serum, but also to some condition of the animal into which serum is injected, inasmuch as such serum which is not hæmolytic for the majority of animals may be so for the minority.

The mortality after injection of serum and virus has been two cases out of twenty-one cases, which is approximately equal to 10 per cent. Both animals had hæmoglobinuria, to which the death must be attributed. The most certain way to produce a reaction under the influence of serum is the simultaneous injection of virus into the jugular vein and the serum under the skin. The quantity of serum varies according to the size of the animal. Its minimum dose has not yet been ascertained. The average dose was 300 cc. The least alarming reaction after injection was obtained when a second injection of serum was made before the rise of temperature began. This is probably the method which will give the best results.

The above investigations which have demonstrated how to confer active immunity against horse-sickness apply only to mules, because I have not had sufficient horses with which to experiment on a large scale. But I feel confident that the one drawback to this method, the occasional occurrence of hæmoglobinuria, can be prevented.

FURTHER REMARKS ON THE CO-RELATION OF SOME SOUTH AFRICAN STOCK DISEASES.

By ALEXANDER EDINGTON, M.D., F.R.S.E., Director of the Government Bacteriological Institute, Grahamstown, South Africa.

THE South African disease known as heartwater, occurring among sheep and goats, seems to have been unknown forty years ago, and is peculiar to South Africa. It is characterised, experimentally, by the difficulty which is experienced in transferring the malady from sick to sound animals. Subcutaneous injections, of quantities so large as 20 cc., of the fresh blood of an animal dying of the malady, almost invariably fail to induce the disease, while doses of 10 cc., injected into the jugular veins, frequently fail if the animal inoculated has come from an absolutely clean area and is itself in perfect health.

Owing to these facts I have been convinced that neither the goat nor the sheep was the proper "host" for the contagion, and that one would eventually find the true disease in some other species of animal.

I have already recounted elsewhere [1] experiments and observations which I had made, from which the following conclusions were drawn :—

1. That the diseases known as horse-sickness, heartwater, impunga, boschziekte, gall-sickness, veldt-sickness, black-lung, and rivierziekte should be regarded as one, or phases of one, malady.

2. That heartwater can be transmitted to cattle.

3. That horse-sickness can be transmitted to cattle.

4. That horse-sickness can be transmitted to goats.

5. That the spontaneously occurring cases of veldt-sickness in cattle present *post-mortem* phenomena which are more or less identical with those of cattle dying from horse-sickness or heartwater (experimentally produced).

6. That cattle born in the Karroo are liable to die when transported to the coast districts, and that this disease is known as impunga or veldt-sickness.

7. That heartwater may be transmitted in a non-virulent form to goats, and, by transmission of blood from goat to goat in a series, can be ultimately raised in virulence.

8. That heartwater blood cannot be preserved in a virulent form, while, conversely, horse-sickness blood can be so preserved for an indefinite period.

9. That the blood of cattle dying of veldt-sickness can produce, by forced infection and subsequent transmission from goat to goat, typical heartwater.

Other conclusions were drawn which need not concern the present paper.

In the experiments which had been made up till that time, success had attended attemps to transmit the disease known as horse-sickness from the horse to goats and cattle, and the disease known as veldt-sickness in cattle to goats, and I had already shown, in my Annual Report for 1898, that heartwater in goats could be transmitted to

[1] Report of Director of Bacteriological Institute, 1902 : "Journal of Hygiene," Vol. III., No. 2, 1903. Proc. S.A. Assoc. for Advancement of Science, 1903.

Karroo cattle by inoculation with the blood of goats dying from heartwater which had been acquired by inoculation from sick goats. This discovery, at first discredited, was corroborated at a later date by other experimenters.

The corrollary to these experiments would have been a demonstration that heartwater could be conveyed to horses. Unfortunately all attempts, even by the intravenous injection of huge doses of heartwater blood, failed to produce anything more than a transient elevation of temperature in the horse.

Eventually concluding that the failure was due to the contagium, in heartwater, being not only relatively altered, but reduced in virulence, I determined to endeavour to exalt its virulence in clean goats by any means which might seem likely to succeed.

Last year the Colonial Veterinary Surgeon, with the assistance of the Colonial Entomologist, produced heartwater in clean goats from infected ticks which had been hatched out at Cape Town. With this strain of blood I inoculated clean goats in this Institute, and from that time have transmitted it in an unbroken series till this date.

I arranged to draw the blood from the infected animals immediately when the temperature was seen to be actually high. While doses of 10 cc., injected intravenously, have formerly been found to be occasionally uncertain, I have found that smaller doses (2 cc.) are absolutely certain if repeated on the subsequent day, and by using 10 cc. doses in this way the blood has been greatly raised in virulence. Under the operation of this method the period of incubation has come to be fairly exact to eight or nine days, while formerly it was irregular and generally longer.

With the blood of a goat dying from this strain I inoculated two horses, A and B, with 20 cc., by subcutaneous injection, on the 18th February. This inoculation was repeated on the subsequent day. Horse A was a clean animal, while B had already been inoculated with less virulent heartwater blood, on the 13th January, without obvious effect.

The temperature of Horse A began to rise on the sixth day, and it died on the morning of the twelfth day. The clinical symptoms were quite in accord with what is usually observed in horse-sickness, and the *post-mortem* revealed changes which are characteristic of that disease. In addition, however, to the ordinary changes, there was a yellowish discolouration of the serous exudates which is somewhat more commonly associated with the disease known as " biliary fever." The purplish congestion of the stomach, which Prof. M'Fadyean has laid so much stress on as being characteristic of horse-sickness, was present in a very definite degree.

Horse B had a definitely elevated temperature on the seventh day, and thereafter continued in fever until the eighteenth day, when the temperature returned to normal.

The goat blood used for these horses was controlled by inoculating it into two clean goats (A and B), which died thereafter on the fourteenth and thirteenth days, respectively, of heartwater.

Horse C was inoculated with 10 cc., by subcutaneous injection, of the fresh blood of Horse A, on the 29th February.

On the sixth day the temperature of Horse C was seen to be definitely rising, and the animal died during the early morning of the

eleventh day. The *post-mortem* examination showed very characteristic signs of horse-sickness.

Horse D was inoculated with 1 cc. of the blood of Horse A, which had meanwhile been preserved in equal parts of glycerine and water to which 1 per 1000 of phenol had been added. This solution is that with which we preserve the virus of horse-sickness.

The inoculation was made on the 12th March by subcutaneous injection. On the seventh day the temperature began to rise, and the animal died on the early morning of the fourteenth day.

The *post-mortem* examination showed the usual signs of horse-sickness, but in this case likewise there was found the somewhat yellowish discolouration of the exudates. Chemical examination has proved this to be due to bile pigment.

Horse B, which recovered after the inoculation, was thereafter inoculated with $\frac{1}{2}$ a cc. of *horse-sickness* blood that had been recently preserved. This blood proved virulent to a clean horse in a dose of 0·075 of a cc. No result followed this inoculation, and the horse must therefore be regarded as having been "salted" against horse-sickness by the inoculation of heartwater blood.

Two goats (C and D) were each inoculated on the 9th and 10th March by intravenous injection with 10 cc. of the fresh blood of Horse C. As no definite elevation of temperature was noticed, they were again inoculated on the 23rd·March with 10 cc. of the fresh blood of Horse D, by intravenous injection.

In the case of Goat C the temperature began to rise during the early morning of the eleventh day, and the animal died of characteristic heartwater on the fifteenth day.

The temperature of Goat D began to rise on the ninth day, and after a severe fever (maximum being 107·8° F.) which lasted until the eighteenth day after the date of the re-inoculation, it returned to normal. On the twenty-third day it was inoculated intravenously with 10 cc. of fresh blood drawn from a goat which was dying of heartwater, and at the same time three clean goats were inoculated with the same blood. All three of the control goats were killed with heartwater, while Goat D remained entirely unaffected.

From these experiments I would, therefore, draw the following conclusions :—

1. That heartwater can be conveyed to the horse.

2. That the conditions produced are identical with horse-sickness.

3. That the contagium becomes relatively altered in the horse, and is then more or less non-virulent for goats after its passage through the former.

4. That the contagium developes in the horse to its highest limit, and, while in that animal, forms hypothetical "resting bodies" which are capable of being preserved indefinitely in the material already described. In this respect the result is in agreement with what we know of horse-sickness.

In all, five horses have been killed with the virus produced by Horse A after its inoculation with heartwater.

The *post-mortem* examinations have revealed in every case phenomena which are characteristic of horse-sickness. In two cases the spleen was larger than normal. This is not usual in horse-sickness, but has occurred, in my experience, in a number of cases.

In all five animals the golden discolouration of the intermuscular exudation was found. In one animal (Horse D) which recently suffered from the infection of piroplasma equi, only two parasites were found after repeated and careful examinations. This horse was one of the two which had an enlarged spleen. In the other cases no parasites were ever found.

Recently I endeavoured to determine whether or not blood which was heavily infected with piroplasma equi could be preserved. For this purpose I withdrew some blood from the jugular vein of a horse which was severely infected and whose blood corpuscles to the extent of nearly 50 per cent. contained parasites. The blood was mixed with the glycerine-water-phenol solution already described. A week later 20 cc. of this blood was injected into the jugular vein of Horse C on the 12th February. No result having followed this inoculation I repeated the injection on the eleventh day. During the subsequent ten days no alteration of temperature was observed, nor were any parasites discovered in the blood of the animal. On the eighteenth day it was inoculated with the blood of Horse A, but no parasites were found at any period up till the death of the animal, nor were any found in the internal organs.

In goats dying of heartwater it is usual to find the gall bladder filled with bile which is generally of a brilliant dark bottle-green colour. The contiguous organs are commonly stained by it of a colour which varies from orange - yellow to dirty green, and the omentum is frequently stained irregularly. It would therefore seem that the action on bile secretion displayed by the contagium in the goat has been carried over to the horse.

In conclusion, I have to express my grateful appreciation of the assistance rendered to me by the Senior Assistant Dr J. M. Coutts, recently acting Assistant-Professor of Bacteriology at University College, London.

EDITORIAL ARTICLE.

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THE POST-MORTEM DIAGNOSIS OF ANTHRAX.

In connection with the subject of anthrax, the question of diagnosis is the one of supreme importance to the practising veterinary surgeon. In a sense that is true of all diseases, but the ability to make a correct diagnosis in suspected cases of anthrax acquires special importance from the fact that an error in either direction may have very regretable consequences. Failure to recognise a case of the disease when it comes under observation generally involves neglect of the special precautions which ought to be taken with the object of guarding against the infection of other animals or of human beings ; and, on

the other hand, to mistake some other disease for anthrax usually means that the owner of the dead animal and the Local Authority are quite needlessly put to a good deal of trouble and expense. To these untoward consequences of errors in diagnosis may be added the fact that when a practitioner makes a mistake there is a risk, greater than in the case of some other diseases, that the blunder may be exposed, to the serious detriment of his professional credit.

Fortunately, in the immense majority of cases of suspected anthrax, the steps necessary to conduct to a correct diagnosis are of a very simple character. At the same time it must be admitted that they are not so simple as was at one time generally believed. What we have in mind in making this reservation is the belief, once very widely prevalent and still held by some, that in suspected cases of anthrax a correct diagnosis may be arrived at by a process as simple as "viewing the body" at a Coroner's inquest. To pronounce a case to be one of anthrax simply because the carcase has rapidly undergone putrefaction, with the consequent escape of blood or blood-stained discharge from the natural orifices of the body, is at the present day absolutely unjustifiable, and, on the part of a veterinary inspector, equivalent to a confession of professional incompetence.

With certain exceptions which will presently be discussed, in the domesticated animals anthrax is a septicæmic disease, in which, before death, the whole volume of blood becomes extensively invaded by the specific bacilli. Such being the case, all that is necessary to arrive at a correct diagnosis in the immense majority of cases, is to ascertain by microscopic examination whether anthrax bacilli are present in the blood or not. When the animal has not been more than a few hours dead, and an invasion of the blood in the peripheral vessels of the body by putrefactive bacteria may therefore be excluded, a mistake in diagnosis is scarcely possible except to the veriest tyro in microscopic work.

But, of course, there are many cases in which the animal has already been dead for from twelve to twenty-four hours before the practitioner has an opportunity to make a microscopic examination of the blood, and there is then always a possibility that even the peripheral blood may contain putrefactive bacilli. Even in these circumstances the diagnosis presents little or no difficulty to the experienced bacteriologist, who is able to distinguish between anthrax bacilli and the morphologically similar microbes that are likely to be present in partially putrid blood. Moreover, we feel justified in asserting that diagnosis is still easy to those who have no claim to be considered experienced bacteriologists, owing to the remarkable reaction which is obtainable by staining anthrax blood with aqueous solution of methylene-blue.[1] For the practitioner, the great value of this reaction lies in the fact that it is much more easily seized with the eye than the

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[1] See this "Journal," Vol. XVI., p. 35 and Vol. XVII., p. 58.

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morphological characters which distinguish anthrax bacilli from malignant œdema bacilli and other putrefactive bacteria, and that, with the exceptions to be hereafter noted, the reaction is obtainable within any period up to twenty-four hours after death.

The main purpose of this article is not to emphasise the ease and certainty with which one may arrive at a correct diagnosis in suspected cases, but to discuss those exceptional circumstances in which microscopical examination cannot be relied upon as the guide, and to consider what is then the proper course of procedure.

It has already been said that in the domesticated animals anthrax is, as a rule, a septicæmic disease, in which, at the time of death, great numbers of the bacilli are present in the blood. It is unlikely that this statement will be disputed by anyone whose experience entitles him to hold any opinion on the subject, but it is probable, and indeed from the recent literature on the subject it is evident, that there is still a good deal of diversity of opinion as to the frequency of the exceptional cases, that is to say, the cases in which at the time of death anthrax bacilli are absent from the blood, or are present in such small numbers as to render microscopic examination an uncertain method of finding them.

As in this country the immense majority of cases of anthrax occur in the ox, it is naturally of most importance to know in what proportion these exceptional cases are to be met with in animals of that species. The opinion which we venture to express on the subject is, that it is not at all certain that cattle ever die from anthrax before a general invasion of the blood by the bacilli has taken place, and that, at any rate, if such cases do occur, they are of extraordinary rarity. It must be admitted that the experience obtainable in this country at the present day does not enable one to accumulate much evidence to show whether cases of anthrax with few or no bacilli in the blood occasionally occur in the ox or not, for, as is well known, suspected carcases are now generally buried or destroyed intact, whether anthrax bacilli have previously been detected in the blood or not, and there is thus little opportunity to discover whether the recorded cases of anthrax would be increased if the spleen and other internal organs were submitted to microscopic examination in every suspected case. However, in this matter the onus of proof lies with those who maintain the unreliability of microscopic examination of fresh blood from a peripheral vein, and in the meantime the practice of pronouncing any case in the ox in which the fresh blood appears to be free from bacilli to be not anthrax appears to be justified.

It can hardly be necessary to say that what has just been stated does not apply to animals that have been slaughtered during the course of an attack of anthrax, for the general blood invasion takes place only a very short time before death when the disease is allowed to run its natural course. Hence, when an animal suffering from

anthrax is killed the bacilli may or may not be detectable by micro-
scopic examination of the peripheral blood, although they would
invariably be found if the examination were extended to the internal
organs.

In this respect the sheep is in the same category with the ox, that
is to say, the apparent absence of the bacilli from the fresh blood,
taken from any part of the body, warrants one in diagnosing the case
as not one of anthrax.

On the other hand, it is a well-established fact that in the pig
failure to detect anthrax bacilli by microscopic examination of the
blood leaves it quite possible that the case was nevertheless one of
anthrax.   Indeed, in that species death occurs in a large proportion
of cases while the blood is still free from the bacilli, or contains these
in such small numbers as to make microscopic examination altogether
unreliable as a means of diagnosis.

In the horse, also, death from anthrax may occur while the bacilli
are still sparsely represented in the blood, but in this species such an
occurrence is much less frequent than in the pig.   In the great
majority of cases of anthrax in the horse blood taken from any of the
peripheral veins immediately after death contains the bacilli in such
numbers that there is no danger of their being overlooked, but on an
average they are less numerous than in the ox, and their apparent
absence on microscopic examination does not justify a decided
opinion.

How, it may now be asked, ought these facts to be recognised in
dealing with suspected cases of anthrax in the domesticated animals ?
Does the fact that microscopic examination of the blood is not in all
cases a reliable method of diagnosing anthrax warrant the veterinary
surgeon in proceeding to make a complete *post-mortem* examination,
and in searching for evidence of the disease in the internal organs,
whenever he is called upon to decide whether a given animal has died
from anthrax or not ?   Needless to say, the answer to that question
must be in the negative.   In the first place, it may be pointed out that
such a procedure is expressly forbidden in the Anthrax Order of 1899,
which says : " In no case shall the skin of the carcase be cut or shall
anything be done to cause the effusion of blood, except by or under
the supervision of a veterinary inspector, and so far as may be
necessary for the purpose of microscopical examination." It might
be supposed that these directions apply only to cases in which the
nature of the disease has previously been established, but, from the
immediately preceding paragraphs it is evident that the sentence just
quoted was intended to apply to the carcases of " suspected," as well
as of " diseased," animals.   In this country, therefore, it is at present
illegal to make an ordinary *post-mortem* examination, or to expose the
internal organs, of any carcase to which a suspicion of anthrax is
attached.   But, if the opinion previously expressed as to the con-

stancy of anthrax bacilli in large numbers in the blood of cattle dead of anthrax is correct, it is obvious that it is permissible to eviscerate the carcase of an animal belonging to either of these species as soon as a proper microscopic examination of the blood has failed to reveal the presence of anthrax bacilli, for such result removes any suspicion that previously existed. However, the latitude which this allows in dealing with the carcases of cattle or sheep supposed to have died from anthrax is more apparent than real, simply because there are still many veterinary inspectors who do not feel competent to base their diagnosis on their own microscopic examination of the blood.

So far as cattle and sheep are concerned, the matter may be summed up by saying that in dealing with suspected carcases it is at once unnecessary and undesirable to make an ordinary *post-mortem* examination—unnecessary, because a microscopic examination of the blood is sufficient to establish the nature of the case, and undesirable, because the evisceration of an anthrax carcase always involves some risk to the operator and necessitates subsequent very thorough disinfection of the place in which the *post-mortem* examination has been made.

In spite of the fact that microscopic examination of the blood is not absolutely reliable for the diagnosis of anthrax in the pig, we believe that in that species also it is seldom necessary to proceed to make an ordinary *post-mortem* examination when the disease is suspected. In the great majority of cases of anthrax in the pig there is a clinical history that hardly leaves any doubt as to the nature of the disease. Inquiry will generally bring to light the fact that part of the recent diet of the pigs has been the raw flesh or entrails of some other farm animal unexpectedly found dead, and if any considerable number of pigs have contracted the disease some of them are almost certain to have displayed more or less swelling in the region of the throat. At least in this country, such a combination of circumstances fully justifies a positive diagnosis of anthrax, although, of course, it is always desirable to make a microscopic examination of the blood of the dead animals.

Unfortunately, in equine anthrax the symptoms and the circumstances connected with the case seldom do more than justify a strong suspicion as to the nature of the disease, but in the majority of cases the bacilli will be readily discoverable in the blood by microscopic examination. We believe that the exceptions are so few as to justify the veterinary surgeon in proceeding to expose the internal organs when microscopic examination of the fresh blood has indicated that no anthrax bacilli are present in it.

The opinion that in suspected cases of anthrax, at least in the ox and sheep, microscopic examination of the blood taken from a peripheral vein is a reliable guide to diagnosis, is not universally accepted,

and we understand that at the present time in Germany the official view is that it is much safer to trust to the result of a cultural examination of the blood.

The evidence cited in support of this view is far from convincing. The practical question is not whether anthrax bacilli can, by the method of culture, be detected in putrid or semi-putrid blood taken from the interior of the body when it is no longer possible to recognise their presence with the microscope, but whether in the ordinary circumstances in which the diagnosis has to be made microscopic examination of blood taken from a peripheral vein, such as that of an ear or a foot, is an untrustworthy method of diagnosis. Here, no doubt, a good deal turns on what is meant by ordinary circumstances. With rare exceptions in this country the suspected carcase is found within twenty-four hours after death, and it may be asserted with much confidence that within that period, at all seasons of the year, microscopic examination of blood from an ear, or a foot, or the tip of the tail (in an ox), is quite reliable. Except during the summer months, blood taken from such a peripheral vessel in the circumstances will, if the case has been one of anthrax, show the characteristic bacilli. Moreover, when methylene-blue is the stain employed the violent reaction is generally obtainable with sufficient distinctness until the third day after death. Finally, this latter reaction is sometimes still recognisable after the anthrax bacilli have entirely disappeared, as proved both by cultural and inoculation experiments.

Reviews.

Annual Reports of Proceedings under the Diseases of Animals Acts, etc., for the year 1903.

In addition to the usual statistical tables, the account of the proceedings under the Contagious Diseases of Animals Acts for the past year comprises reports by the Chief Veterinary Officer and the Assistant Secretary to the Board of Agriculture and Fisheries. Agriculturists have reason to congratulate themselves that neither of these has had to record anything very startling in connection with the incidence of the contagious diseases of animals during the year 1903.

In the introductory part of his report the Chief Veterinary Officer takes the opportunity to impress upon stock-owners the advantages which they now enjoy in consequence of the success which has attended the efforts of the Board to stamp out some of the animal plagues which are still the cause of great loss in most foreign countries. To the former triumphs of the Board in this direction the past year has added still another success, for the report has to chronicle the pleasing fact that for the first time in the history of the

disease no case of rabies was detected in Great Britain during the twelve months under review.

Unfortunately, the report has to take notice of other facts which are of a less satisfactory character. The outbreaks of glanders reported during the past year were 25 per cent. in excess of those for 1902, and there was also a disquieting increase in the prevalence of anthrax. In connection with the former disease, the Chief Veterinary Officer raises, but does not attempt to answer, the question whether the increase in the number of cases reported is due to an actual spread of the disease, or to a more frequent resort to mallein, resulting in the detection of cases that would formerly have been overlooked. In view of the fact that during the year the average number of cases in each outbreak was less than two, the latter explanation can hardly be the correct one, for it can scarcely be doubted that if mallein were systematically employed in infected stables, and the animals reacting to the test were dealt with as glandered, the ratio between the number of outbreaks and the number of horses found to be diseased would be very different from what it has been hitherto.

The report by the Assistant Secretary covers much the same ground as that by the Chief Veterinary Officer. Contrary to what might have been expected, it deals not only with what might be called matters of administration, but also with questions which a layman can hardly pretend to discuss with authority. For example, what is said under the head of anthrax is mainly a defence of cremation, as a method of disposing of anthrax carcases superior to burial, and we find the Assistant Secretary laying down the law to the effect that " it is undoubtedly the case that destruction by fire or by exposure to a high temperature is the most effectual manner of disposing of such carcases, providing that this can be done without cutting the carcase." Surely this is a matter requiring for its decision some knowledge of the bacteriology of anthrax, and with all humility it may be suggested that prompt burial is an effectual method of disposing of anthrax carcases, and, where practicable, not inferior to cremation. In discussing the relative merits of the two methods, the Assistant Secretary reminds Local Authorities that, in order to comply with the provisions of the Anthrax Order, about two tons of lime ought to be used in burying an anthrax carcase, whereas inquiries made by the inspectors of the Board elicited the fact that in many cases considerably less than half a ton is used. This, it may be observed, is a fact that has not the least relevance to the question whether cremation is superior to burial, simply because there are no sufficient grounds for believing that a carcase buried with lime, whether the amount be two tons or half a ton, is any safer than one buried without any lime at all. The facts connected with the incidence of anthrax in this country are wholly inconsistent with the view that the increased prevalence of the disease during recent years is in any important degree due to the circumstance that carcases have generally been disposed of by burial, and disappointment is in store for those Local Authorities who, at the instigation of the Board of Agriculture, have erected crematoria for the destruction of anthrax carcases, in the expectation that a reduction in the prevalence of the disease in their districts will thereby be effected.

Report of Proceedings under the Diseases of Animals Acts for the year 1903. Department of Agriculture and Technical Instruction for Ireland.

THE case of Ireland is probably the most striking illustration to be found anywhere of the advantage of an insular position to a country in which the breeding and rearing of animals forms an important part of the agricultural industry. With regard to the incidence of contagious diseases among its

domesticated animals Ireland would appear to be at the present moment the healthiest country in the world, and it undoubtedly owes its good fortune in this respect mainly to the circumstance that it is an island with a very restricted import trade in animals. In the second line it owes it to a vigorous and intelligent administration of the Acts relating to the contagious diseases of animals.

The present report shows that among a total of over 11,000,000 of domesticated animals the outbreaks of contagious disease during the past year occurred only in the proportion of one outbreak for every 11,000 animals. Some of the facts recorded in the report are even more striking than this. Thus it appears that among a total of nearly 5,000,000 cattle only one of the diseases scheduled under the Contagious Diseases of Animals Acts occurred during the year, viz., anthrax, and of that disease only four outbreaks with eleven animals attacked were reported. The outbreaks of glanders were five, and of swine-fever 175. The two most prevalent diseases were sheep-scab, with 655 outbreaks, and parasitic mange among horses, with 195 outbreaks. In both of these, and also in the case of swine-fever, the figures for the past year are, unfortunately, slightly in excess of those for 1902. During the year two cases of rabies occurred, a fact that is somewhat remarkable seeing that no case of that disease was detected during the previous year.

A Treatise on Epizootic Lymphangitis. By Captain W. A. Pallin, F.R.C.V.S., Army Veterinary Department. Williams & Norgate, London, 1904.

THIS little book appears opportunely in view of the danger that the disease of which it treats has already established itself on a permanent footing in this country. Captain Pallin appears to have had considerable experience in connection with the disease during his period of service in India and China, and can therefore speak at first hand regarding the clinical aspects of the affection. He was also provided with a number of excellent photographs showing the cutaneous and other lesions, and these have been reproduced to illustrate the book. But the author has naturally desired to render his account of the disease as complete as possible, and has therefore drawn freely from the previously published literature on the subject. In this connection he acknowledges his indebtedness to Tokishige's monograph, and to the article on epizootic lymphangitis in the text-book of Nocard & Leclainche. Candour compels us to say that the author appears to have adhered with remarkable fidelity to the account of the disease which is given in these two articles, and the result is likely to be a little disappointing to those who are already acquainted with these sources of information on the subject. On the subject of staining the author develops more than his usual originality, and gives quite an embarrassing choice of methods by which the cryptococcus may be stained. We fear that Captain Pallin has not made a serious study of the relative merits of the stains mentioned, for he appears to accord the preference to the Gram-Nicolle and Gram-Weigert-Kuhne methods, neither of which is equal to the method of Claudius in simplicity and effectiveness. It is a little unfortunate also that, in mentioning the Claudius method, the author has omitted to accord to Mr Bowhill the credit which is due to him for having been the first to point out the superiority of this stain over all others for staining the cryptococcus.

On the question of differential diagnosis too much importance appears to be attached to a number of points, such as the presence or absence of fever, the general condition of the patient, the colour and consistency of the pus, and the situation and macroscopic characters of the lesions. Here again the author seems to have followed too closely the earlier accounts of the disease,

the majority of which would appear to have been written by observers who had not had much experience in connection with glanders and farcy. With regard to the differential diagnosis of the latter disease and epizootic lymphangitis, nearly all that is useful to the practitioner may be summed up by saying—(1) that no case can be pronounced to be one of epizootic lymphangitis until the cryptococcus has been discovered in the lesions, and (2) that in an infected locality no horse can be pronounced free from glanders unless it has been tested with mallein and has failed to react. Fortunately, owing to the readiness with which the cryptococcus may be demonstrated, no problem in diagnosis is easier than the recognition of epizootic lymphangitis when external lesions are present.

The prophylactic measures which Captain Pallin recommends are very complete, and this part of the subject has evidently been carefully thought out. We hear, however, that similar measures have been practised with very indifferent results during the past year or more in various studs of army horses, no doubt because the disease has in many cases a very long period of incubation, during which diagnosis is impossible. From a national point of view, the most important prophylactic measure at the present time would be to forbid the sale of cast horses from the army, or at least from all studs in which any case of the disease has recently occurred.

CLINICAL ARTICLES.

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INTUSSUSCEPTION OF THE SMALL BOWEL IN THE HORSE.

By E. E. MARTIN, Captain, Army Veterinary Department, Army Veterinary School, Aldershot.

A FEW months ago a case was brought to my notice which, as it bears on the interesting subject of Intussusception, appears to me to be worthy of record.

The patient was a Government remount, and was under the care of Mr Sutton C.V.S., to whom I am indebted for the following clinical notes.

The remount was admitted to hospital for catarrh a few days after purchase. After a few days the temperature rose considerably, some difficulty in breathing was seen, and auscultation showed that the lower portions of both lungs were dull and oppressed. From this time onwards until the time of death the symptoms increased in severity. Temperature remained above 103° and under 104°. Appetite practically nil, occasionally a little green grass would be taken. Generally there was a rusty discharge from the nostrils, and on two occasions there was considerable bleeding. Three days before death diarrhœa set in. At no time during the illness was he seen to lie down or show any symptoms of abdominal pain. The total duration of the illness was three weeks.

Post-mortem examination showed the large bowels strikingly

empty, the small intestines 6 feet from the pyloric end of the stomach showed an intussusception, the stomach had a large rent 6 or 7 inches in length in it, and 1½ lbs of ingesta were found in a kind of sac formed by the omentum. Except this accumulation in the omental sac, no ingesta were found in the peritoneal cavity, and there was no general peritonitis. The edges of the rent in the stomach were much thickened, and the pouch of the omentum in which the ingesta lay was considerably inflamed. Both lungs showed well marked pneumonia, the lower portions of each being consolidated.

Mr Sutton, who had made the *post-mortem*, excised the affected parts and sent them to me for inspection. On close examination I found the intussuscepted portion (the intussusceptum) measured 4 inches in length. The peritoneal surfaces of the intussusceptum had become firmly adherent to each other ; indeed, so firm was this union that, when I tried to draw back the intussuscepted portion, the wall of the bowel not involved tore before I could reduce the intussusception. The lumen of the bowel had not been entirely occluded, for I could, with a little squeezing, pass my finger through the intussusceptum. The whole of the mucous membrane of intussusceptum was in a necrotic condition, but the slough was still firmly adherent to the underlying tissue.

The rupture in the stomach was 7 inches in length ; the rupture in the muscular and serous coat was considerably larger than that in the mucous, and gave the idea that the external coats (muscular and serous) had just given way some time before the mucous. The edges of the mucous coat were thickened and were covered in parts by clotted blood. The food that had escaped from the stomach all lay in a kind of sac formed by the omentum, and I could see no signs of any general peritonitis. The portions of the lung forwarded to me were consolidated.

The *post-mortem* examination thus revealed three gross lesions, viz. :—

1. Pneumonia.
2. An intussusception of the small intestine.
3. A ruptured stomach.

The question what relations these lesions had one to another appears to me a very interesting one.

I think the pneumonia was the original trouble, and from its onset the animal's illness may be dated. Between the pneumonia and the abdominal lesions I do not think we can make any close connection. The most important question, however, is what was the date of the intussusception, and what relation does that bear to the ruptured stomach. Judging by the firm union between the serous surfaces of the intussusceptum, the condition of the mucous membrane, and the entire loss of appetite which was shown in the later stages of the illness (and which in my opinion the pneumonia alone would not account for), I should think the intussusception must have been in existence at least a week, if not more. Probably at first the lumen of the bowel was not quite blocked for passage of food ; when it did become practically blocked nature probably made a violent effort to remove the obstruction, and those efforts would express themselves in the diarrhœa, and most probably in attempts at vomition. It may be said that no vomition or attempts at vomition were ever seen, and

such was the case, but it must be remembered that the animal was not by any means constantly watched. The reasons I think attempted vomition must have taken place, and that it was due to this that the stomach was ruptured, are as follows: (1) Vomition is one of the most constant symptoms of intussusception of the small intestine or any complete obstruction there. (2) The close relationship that always exists between ruptured stomach and attempts at vomition in the horse. (3) The entire absence of any other cause for the rupture, such as over-distension from food or work on a full stomach.

With regard to (1), that vomition is one of the constant symptoms of intussusception, I would like to refer to a case only recently reported by Pickering. In the Veterinary Record, Vol. XV., page 305, he recounts a case the outlines of which are as follows :—" Gelding attacked during the night with acute symptoms of abdominal pain followed by vomiting. *Post-mortem* showed distended stomach and intussusception of duodenum 2 feet from the pylorus."

I think it very probable that the rupture of the stomach occurred about the same period as the onset of the diarrhœa, which was three days before death. Directly the stomach was ruptured and the ingesta escaped the attempt at vomition would probably cease.

The striking clinical feature of the case was that the horse never showed any signs of acute abdominal pain. We are accustomed to associate ruptured stomach with a rapid and extremely painful death. Death usually is due to an acute general peritonitis set up by the escape of the ingesta into the peritoneal cavity and the consequent absorption of toxins. In this case, owing to a peculiar disposition of the omentum, there was no general escape of ingesta into the peritoneal cavity, and this probably accounts for the absence of urgent symptoms.

SEVERANCE OF THE VAS DEFERENS AS A SUBSTITUTE FOR CASTRATION IN THE HORSE.

By J. TAGG, M.R.C.V.S., Croydon.

AS castration of the horse is a subject about which much has been written, I feel rather diffident at approaching it, but I think that any new method which renders an operation less painful and more scientific, and precludes the possibility of the ubiquitous empiric from indulging in it, should be employed by us if practicable.

I believe that it is frequently noticed among the natives of India that when the vasa deferentia, either from any diseased cause or traumatic injury, become occluded, atrophic changes occur in the testicle. Accepting as a physiological truism, that demand governs the supply of any organ, it occurred to me that by excising a portion of the vas deferens, and so closing all connection between the gland and the vesicula seminalis, such atrophic changes might be brought about as would obviate the necessity for castration. I believe that in mammals which have vesiculæ seminales the amount of semen stored within them has a direct ratio to the desire. In the gelding

the vesiculæ gradually atrophy after castration, and the same changes would naturally occur after excision of the vasa deferentia. The same changes no doubt occur after the barbarous practice of "mulling" resorted to by the natives of India with cattle. They simply mull or rub the spermatic cord and testicle with one smooth stone over another, thereby destroying the organ.

The operation of excising the vas deferens could not be recommended for racing stock, in case the question might arise as to whether they were castrated. I would certainly recommend it with all other animals, as it struck me that those operated upon showed much better crests and altogether better fronts than those operated upon in the usual manner, for the simple reason, I suppose, that they retained for a longer period the nature of the male.

As I had some yearlings running in the paddocks I thought it a good opportunity to try the operation. After they recovered from the operation I kept them under observation for exactly a year, running about with fillies in the paddocks, and behaving just as if they had been castrated in the ordinary way. One can always distinguish a slight swelling of the scrotum a year after the operation. About six months after the testicles feel like a soft india-rubber ball.

CASE I. *Subject*—bay country-bred thoroughbred colt (yearling). *Anæsthetic*—chloroform used with a Carlisle muzzle.

The colt was thrown and chloroformed, and the off hind leg drawn forward as for castration. I tried to secure the testicle so as to obviate its slipping by placing the clam over it, but this I found did not answer. I then passed a suture needle through the testicle, much in the same way as one does through a tumour for dissection. An incision was now made about two inches above the testicle, over the cord, but posteriorly, so as to avoid any accident with the artery.

By incising well behind one cannot avoid finding the vas deferens, which shows up from its white colour, and feels hard to the touch, very like a large bundle of nerve fibres. About an inch of the vas deferens was cleared, and the duct was ligatured with catgut at two places three-quarters of an inch apart. The duct was then divided between the two ligatures. If the proximal end is not ligatured there will be a constant discharge of semen for some time after the operation. This I found in the case of a pony I operated upon for a poor native. The skin was sutured, and iodoform dusted over it. The dressing used was corrosive sublimate lotion, 1 per 1000.

After the operation there was great difficulty in rousing him, and he completely lost all command over his legs for about half an hour after being released. He, however, did well, and after keeping him in a loose-box for three weeks I turned him out with others in the paddocks.

CASE II. *Subject*—a roan country-bred colt (yearling). *Anæsthetic*—chloroform used with a Carlisle muzzle.

Operation was performed as in Case I. I found I had cut rather too low down, and was only just on the globus minor, but rather than make a larger incision I incised at this point. I did not suture the incision, as I had handled the parts a good deal, and I imagined there were chances of its suppurating. The dressing used was chloride of zinc lotion, 1 in 16.

The subject was kept in a loose-box for a fortnight, and afterwards turned out in the paddocks with others.

CASE III. *Subject*—a bay cross-bred stallion, aged three years.

The animal having been cast as before, cocaine was injected into the cord, about 1½ grs. I found that the operation is not so easy without chloroform, owing to the action of the cremaster muscle. The assistant has to exert too much traction on the testicle to keep the cord low enough for operation. I would therefore always advise chloroform, as it is so much easier to operate when everything is in a state of inertia. The incision was sutured with carbolised catgut and dressed with chloride of zinc lotion, 1 in 16.

This subject had previously given my blacksmith a good deal of trouble. He frequently broke loose and rampaged about the bazaar after mares.

After the operation he became quiet quite, and worked and behaved exactly like a gelding.

I performed the operation on three other subjects, but afterwards lost sight of them.

I have tried to operate from one incision, but I found I did very much better by incising both the cords. The scrotal coats seem to get in the operator's way, and this necessitates too much handling of the parts, which ought to be avoided ; besides, I found it very much lengthened the operation.

SUTURE OF THE DIVIDED RECURRENT TO THE VAGUS IN ROARING.

By the Same.

IN 1896 I recorded a successful case of a mare I operated upon, and then promised to record others, which promise, although late, I beg now to fulfil.

CASE I. 18th February 1897. *Subject.*—Thoroughbred Waler gelding, "Spes," a steeplechaser.

Cast in the usual way and chloroformed, using a Carlisle muzzle. The seat of operation was shaved and washed with carbolic lotion, 1 in 20.

The recurrent nerve was found resting on the œsophagus, and the vagus on the upper and inner border of the carotid. The former nerve was lightly sutured to the latter with carbolised catgut. The muscles and skin were sutured—the former with carbolised catgut, and the latter with flax thread. The dressing used was Lister gauze and iodoform.

The subject was discharged after a month.

About four months later he was so much improved, as far as the roaring was concerned, that they put him into fast work. He was never able to win races, so got sold, and we lost sight of him.

This horse roared so badly before the operation that I had some doubts about chloroforming him, and when on his side after being thrown he roared so badly, and appeared so distressed, that I was afraid he would collapse altogether. He actually roared at a slow trot, yet he so far recovered as to be put into fast work.

CASE II.   28th February 1897.   *Subject.*—Thoroughbred Waler, "Banshee," won jumping competition at Tollygunge, Calcutta, 1897. Roared badly.

In this case I operated lower down, over the lower third of the jugular furrow, directly under the jugular vein and at the upper border of the sterno-maxillaris.   The recurrent nerve was found resting on the œsophagus and the vagus on the upper and inner border of the carotid.   There was a good deal of hæmorrhage owing to an affluent of the jugular getting cut.   Muscles and skin sutured as before.

I discharged the subject after keeping him in my hospital for a month.

Unfortunately, some three months afterwards he picked up a nail while out at exercise, and eventually succumbed to tetanus, so I am unable to record whether the operation was a success or not.

CASE III.   28th February 1897.   *Subject.*—Bay Waler gelding (carriage horse).   Roared badly.

In this case there was very little hæmorrhage after the skin was incised.   If one be careful the large trunks of veins can be easily avoided, thereby ensuring the finding of the inferior laryngeal nerve, which was in this case found by the side of the œsophagus.   The vagus was rather low down, on the inner side of the carotid.

This case was so much improved by the operation that his owner drove him for two years afterwards.   Before operation he roared so badly that he could not be used.

CASE IV.   I have no notes of this case, but will state the facts as far as I recollect them.

Sometime in 1897.   *Subject.*—Bay English coach horse, " Caractacus."   He roared so badly that we could no longer use him in the carriage.

I performed tracheotomy on him in 1896, but owing to his rubbing the tube during the rains and setting up a growth on the trachea, I performed the new operation on him.   He improved so much about six months after the operation that he was sent back to the coach-house stable, and he was driven to my knowledge for two years afterwards.

CASE V.   This is also a case of which I have no notes, but, owing to certain peculiar circumstances of the case, I can recollect the details of it.

Subject.—A grey Waler mare which had been given to her present owner by a friend of mine on condition that I was allowed to perform the operation on her as an experiment.

The mare roared at a slow trot, but, being such a good-looking one, she was thought worth the risk.   This was the worst roarer I had ever heard.

I operated upon her, and four months afterwards I rode her over a course and found she still roared, but had improved vastly.

About a year later I rode her for 7 miles at a smart canter, and she only just made a slight noise.   At any rate, she became a useful hack, whereas before she was absolutely useless.

For the minute details of the operation I beg to refer readers to the *Journal of Comparative Pathology*, Vol. IX., p. 34.

SYMPTOMS OF INTESTINAL OBSTRUCTION—
AN UNUSUAL CAUSE.

By ROBERT G. ANDERSON, M.R.C.V.S. Wellington, Somerset.

Subject.—A ten years old cart mare, in good condition, and in foal with one month to go.   On the morning of 11th May last, she showed signs of abdominal pain, regarded as due to obstruction from intestinal impaction.   There was no previous history of colic except for one slight attack four years ago.

Treatment.—Stimulants, nerve tonics in milk and linseed tea ; aided by clysters and doses of ol. ricini.   This treatment was supplemented by hot water fomentations to the abdomen.   Chloral hydrate was given at long intervals when the pain seemed greater, to conserve strength.   Caution in the matter of physic was observed on account of the pregnant state of the mare.   Later it seemed as if this caution was to cost the life of both mare and foal, and, seeming to be necessary, aloes was given in a moderate dose.

Death occurred forty-eight hours after the case was first seen.

Peculiarities noticeable had been the soft nature of the bowel contents on evacuation by hand, and evidence on auscultation of general free motion of fluid in the bowels from twelve hours after oil was given, although in twenty-four hours no passage had been obtained.

The absence of evidence of impacted colon on manual exploration was not regarded as proof that such did not exist somewhere in its course ; this indeed was the opinion most inclined to, but the peculiarities mentioned prevented that opinion being held without reserve.   A calculus was not suspected.

Post-mortem examination showed a constricted condition of the terminal portion of the ileum, so that two fingers close pressed together could with difficulty be passed through.   Loosely wedged into the narrowing part anterior to the constriction was a ball-like mass of loosely coiled worms—ascarides.   These, I take it, had been detached from the walls of the small intestine under the action of medicines given, and passed backwards to the constricted part, where ebb and flow had caused their balling.

The cause of constriction may have been the long continued irritation from parasites adherent here, but none were found in the large bowels, nor had any been seen to pass.

𝔄bstracts and 𝔑eports.

AN ENZOOTY OF ACUTE STREPTOCOCCIC MAMMITIS.

By P. Dubois.

NUMEROUS investigations into the nature of mammitis in milch cows have shown that the disease may be produced by one of a number of organisms, and that any given infection is usually due to a single species, rarely to more than one, but that there is no really specific organism except in the case of contagious catarrhal mammitis, which results from infection with a well-recognised streptococcus.

Mammitis assumes an acute or chronic form, and occurs sporadically or enzootically according to the virulence of the infecting organism and the receptivity of the subject.

The contagious forms are of special importance on account of the losses to which they may give rise.

Diekerhoff saw 321 cases between 1873 and 1877. He considers infection occurs by the transmission of organisms from the litter by means of the teat, and has proved that milkers may convey the disease from one subject to another.

In 1877 Zürn saw 140 cows out of a total of 180 attacked by a disease due to a mixture of micrococci, mycotrix, mucor mucedo, and cells resembling those of yeast. The disease was transmissible to ewes and sows.

As recently as 1898 Thiele reported an outbreak among ninety-three cows, of which forty-eight were affected and seven died. The disease broke out suddenly and produced grave general symptoms. The milk became serous, purulent, blood-stained, and of offensive odour. Medical treatment proved unsuccessful. Thiele found strict isolation the only effective treatment.

Owing to the difficulty in certain cases of identifying the different varieties of streptococci, one cannot absolutely declare that the acute and chronic forms of streptococcic mammitis are not due to the same agent, but cases of acute contagious mammitis occur in which the organisms have such well-marked characteristics as to admit of differentiation, and it seems certain that, apart from the organism of *Gelber Galt*, an essentially different variety of streptococcus exists, capable of producing acute contagious mammitis. Dubois has studied an outbreak of acute mammitis due to streptococcus conglomeratus (Kurth).

The disease suddenly attacked a newly-bought cow in a byre of twenty-eight cows on the 10th March 1903. Food was entirely refused, the animal was dull, the temperature rose to 41·3° C., and the mammary gland was tense, hot, and painful. The milk was greyish in colour, and contained grumous fragments. The microscope revealed large numbers of streptococci. Acetanilide internally, iodide of potassium ointment locally, and injection of 3 per cent. boric solution into the gland after milking, proved ineffectual. Another cow standing next the first was similarly attacked a few days later. Ten others were successively affected despite rigorous disinfection and isolation. In two months and a half, however, the disease seemed to have worn itself out.

Fifteen cows had been attacked, and in five the function of the udder was entirely lost; these were slaughtered. In two half the udder was affected;

those last attacked still gave milk from all four quarters, but in much diminished quantity. The streptococcus was obtained in pure culture from the milk and showed the following characteristics : in twenty-four hours it gave very rich cultures in glycerinated bouillon ; the fluid remained clear and the growth was deposited on the sides and bottom of the tube. Microscopically the growth assumed the form of very long chains. The streptococcus grew very slowly on agar and gelatine, gave little white colonies, and did not liquefy the gelatine. No growth on potato.

The streptococcus stained well with simple colours like Ziehl, carbolated thionine, and phenolsafranine. It took Gram well. Two cc. injected intro-peritoneally killed guinea - pigs in twenty-four hours and rabbits in eight days.

The prognosis in these cases is very grave, and when the entire udder is affected the animal should be sent to the butcher. Medical treatment is of little value, although one severe case was cured in three days by frequent effusions of cold water to the udder and by milking at short intervals.

Prophylaxis is the only reliable means of dealing with the disease. The hands of the milker and the cow's udder and teats should be washed before each milking with 2 per cent. carbolic solution. The affected cows should be milked last. Diseased animals should be isolated. The milk should not be spilt in the stable or poured down drains. The litter should be removed and burned and the byre disinfected. The udders of newly bought cows should be examined, and new purchases should be quarantined for some days. Quarantine is very important. Although no milk is being secreted the gland may be infected, and cows with dry udders should therefore be strictly quarantined until after calving.

The danger was well illustrated in the case of two cows sold from the byre in which this outbreak occurred. Being "dry," they did not develop signs of disease but were nevertheless infected, and soon after calving in their new surroundings they became severely attacked. A fresh outbreak would probably have occurred in the premises of the new owner had very rigorous measures of isolation and disinfection not been at once enforced (*Revue Vétérinaire*, 1st Dec., 1903, p. 789).

THE RELATIONS OF AVIAN AND MAMMALIAN TUBERCULOSIS.

DRS. Weber and Bofinger, who have carried out an extensive series of experiments and observations with regard to avian tuberculosis, summarise as follows the results at which they arrived.

In contrast with the bacillus of mammalian tuberculosis, that of avian tuberculosis produces on solid media a moist, slimy growth. When floated on fluid nutritive media it seldom yields a surface growth like the bacillus of mammalian tuberculosis, but grows in the form of little granules and crumb-like masses on the bottom and walls of the flask.

The above were the appearances yielded by nine of the eleven specimens studied by the authors.

Some specimens, however, grow like the bacillus of mammalian tuberculosis, producing on solid media a dry crumpled skin, and on fluid media a superficial growth, the latter especially being indistinguishable from exuberant growths of mammalian tubercle bacilli. This occurred in two cases among the eleven studied. One specimen had been cultivated for years on artificial media, the other had been obtained freshly from the body of a fowl in the form of pure culture.

The specimens yielding growths resembling those of mammalian tuberculosis have been regarded by supporters of the identity theory as transition forms between avian and mammalian tuberculosis. Were this view correct, however, the resemblance should also be borne out by their pathological peculiarities, that is to say, the specimens whose growths resembled those of mammalian tuberculosis should be less virulent for fowls and more virulent for guinea-pigs than those which yielded typical growths. Such, however, was not the case; the two specimens which produced growths most closely resembling those of mammalian tuberculosis proved least virulent for guinea-pigs, and in the authors' opinion produced far less pathological disturbance than the bacilli of avian tuberculosis usually do when inoculated into guinea-pigs.

It is therefore incorrect to view such variations in the method of growth as indicating transition forms between the two kinds of tuberculosis. True transition forms have not yet been discovered.

Moreover, it seems remarkable that a pathological organism so deadly for fowls as is the bacillus of avian tuberculosis should when artificially inoculated (by any other method than intravenous injection) in general prove so difficult to convey to the fowl. (Intraperitoneal and intramuscular inoculation of fowls only proved successful when large quantities of the bacilli were employed.)

On the other hand, in almost every case fowls were rendered tuberculous by once feeding with a relatively small amount of the bacilli. The quantity of bacilli required to ensure infection from the digestive tract must in fact be regarded as very small if we consider that only a portion of the bacilli administered are retained in the body, and that the greater part is again voided with the fæces.

The results of these experiments render it clear that under natural conditions fowls are inoculated through the bowel in consequence of picking up tubercle bacilli passed with the fæces by diseased fowls. The congenital conveyance of avian tuberculosis can in comparison only play a trifling part.

The fact that no tubercle bacilli are found in the fæces of severely infected fowls or such as have died from tuberculosis, cannot be regarded as disproving infection by the bowel. According to the authors' investigations, the excretion of tubercle bacilli occurs in an early stage of the disease. It begins when the tubercles in the Peyer's patches and the solitary glands undergo ulceration. At this time, as the authors have repeatedly observed, fragments of tissue containing large quantities of tubercle bacilli are passed. As in many cases more or less complete healing of the ulcer occurs, excretion of tubercle bacilli with the fæces may in later stages of the disease entirely cease, or at least may occur only to a very trifling extent.

The large nodules projecting beneath the serous coat of the bowel, which are seen in the later stages of the disease, and which at first sight often appear to have no connection with the mucous membrane, have also originated from ulcers in the bowel, usually from ulcerated Peyer's patches which, after destruction of the thin muscular layer covering the Peyer's patch, have been cut off from the rest of the bowel wall by contraction of the strongly developed muscular tissue surrounding the patch. Even at a later stage a fine fistulous opening may often be discovered, through the medium of which they communicate with the lumen of the bowel.

With this extreme sensitiveness of fowls to infection with avian tubercle bacilli through the medium of the bowel, the complete failure of all feeding experiments with much greater quantities of human and bovine tubercle bacilli stands in marked contrast. Until now all attempts to convey mammalian tuberculosis to the fowl have failed.

The bacillus of avian tuberculosis is pathological not only for fowls but also for rabbits, mice, and in a less degree for guinea-pigs. In rabbits it produces

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tuberculosis, and in mice a disease resembling leprosy of man, but in guinea-pigs only suppuration, limited to the point of entrance and the neighbouring glands.

The same pathological organism, therefore, produces entirely different forms of disease in different animals.

Like fowls, rabbits are more readily infected by feeding than by sub-cutaneous inoculation. The frequent occurrence of tuberculosis of the joints and tendon sheaths in these animals is also noteworthy.

When inoculated into mice, the bacilli of avian tuberculosis multiply to an enormous extent without the slightest signs of intoxication or any noteworthy reaction on the part of the tissues. The bacilli are found within the cells, which are mostly newly-formed and of an epithelioid character, less frequently those of normal tissue. The cells are absolutely crammed with bacilli. When infected through the medium of the bowel, mice develop avian tuberculosis in one year; after subcutaneous inoculation with one dose of pure culture, in six months; and after intraperitoneal inoculation, in two to four months.

The bacillus of avian tuberculosis also developes in guinea-pigs. Develop-ment, however, in most cases is of a limited character and leads to the formation of localised abscesses. After subcutaneous inoculation in the region of the abdomen abscesses develop at the point of inoculation and in the inguinal or axillary glands; when the bacilli are given with food, in the follicles of the bowel and in the mesenteric and submaxillary glands. These abscesses usually heal. The bacilli may, however, multiply to a greater extent at the point of entry and in the internal organs even after infection through the digestive tract. In such cases the animals die from the effects of toxins produced by the avian tubercle bacilli; true tuberculosis never develops.

In feeding experiments on rabbits, guinea-pigs, and mice the tubercle bacilli enter the body both through the mucous membrane of the mouth and pharynx and through that of the bowel, making their way firstly into the follicles of the bowel and the mesenteric and submaxillary glands.

The slow progress of avian tuberculosis when inoculated into mice offered a favourable opportunity for studying the paths of infection by which the organisms spread from the digestive tract.

In mice, as above mentioned, the bacilli of avian tuberculosis made their way, firstly into the follicles of the bowel and the mesenteric and submaxillary glands. In seven weeks they had attained the lungs and spleen, in three months the bronchial glands, in four the axillary glands, in seven the inguinal glands, and in nine months the liver and kidneys.

In all cases the lungs were attacked very early and before the bronchial glands. The latter were infected from the lungs themselves. It seems impossible that the lungs can have become infected otherwise than through the thoracic duct in consequence of antecedent disease of the mesenteric glands.

In the case of rabbits fed with avian tubercle bacilli the lungs were again the first of the internal organs to become diseased.

In contrast with this the lungs of fowls themselves either remained quite exempt from disease, or only contained a few nodules.

In these animals the liver was the organ first and most severely attacked. It is clear that the lungs of the fowl itself do not offer favourable conditions for the development of the bacilli of avian tuberculosis.

In cases of infection from the bowel the organs first and most severely attacked are those which prove most liable to disease after inoculation and spontaneous infection, that is, in mice the lungs, in rabbits the lungs and kidneys, and in fowls the liver and spleen.

The bacilli of avian tuberculosis retain their original pathological properties after passage through the bodies of mammals. The authors' experiments

showed that after growth for one to two years in the bodies of guinea-pigs and mice the virulence for guinea-pigs had neither increased nor that for fowls decreased.

In accordance with the statements of French authors, Weber and Bofinger were able to produce typical cultures of mammalian tubercle bacilli from the organs of a tuberculous parrot. On the other hand, they also succeeded in obtaining from the caseated mesenteric glands of a young pig, which showed no other signs of tuberculosis, a typical culture of the bacillus of fowl tuberculosis.

Under natural conditions, therefore, neither had the bacilli of mammalian tuberculosis undergone conversion in the body of a bird into those of avian tuberculosis, nor had the bacilli of avian tuberculosis in the body of a mammal become converted into those of mammalian tuberculosis. (*Tuberkulose Arbeiten, aus dem Kaiserlichen Gesundheitsamte*, 1904, Heft 1., p. 148.)

ON THE PRESENCE OF TUBERCLE BACILLI IN THE MILK OF COWS WHICH ONLY REACT TO TUBER-CULIN.

By STENSTRÖM.

STENSTRÖM first recounts the most important works referring to the above question, and particularly the more recent investigations of Rabinowitsch, Kempner, and Ostertag.

Rabinowitsch and Kempner found, in animals which reacted to tuberculin but showed no clinical appearances of tuberculosis, that the milk occasionally contained tubercle bacilli.

On the contrary, Ostertag, who also experimented with the milk of cows which reacted to tuberculin but showed no clinical signs of tuberculosis, never found tubercle bacilli.

Ostertag, who has investigated this question very thoroughly, in a large number of animals made experiments by feeding calves and pigs with the milk of cows which had reacted. In these cases also he was unable to discover any infection with tubercle bacilli.

Stenström's experiments were carried out in the bacteriological laboratory of the " Separator " Company in Hamra, on cows intended for slaughter. As far as possible antiseptic precautions were observed. The milk was withdrawn into sterile vessels, passed through a centrifugal apparatus rotating at the rate of 3000 to 5000 turns per minute, and the deposit so obtained was mixed with the cream and used to inoculate rabbits intraperitoneally.

In some cases rabbits and guinea-pigs were simultaneously inoculated. Fifty cows were so tested, sixty-eight rabbits and fifteen guinea-pigs being inoculated. The cows had all reacted to tuberculin; several had shown clinical symptoms of tuberculosis, and on *post-mortem* examination revealed somewhat well-developed changes. One rabbit died from peritonitis. The other experimental animals died or were killed within two to five months after inoculation. None of them proved to be infected with tuberculosis. (*Zeits. f. Fleisch and Milchhygiene*, May 1904, p. 277, and *Rev. generale du lait*, 1902, No. 21.)

THE IMPORTANCE OF UROSCOPY IN VETERINARY MEDICINE.

By H. JAKOB.

THE complete examination of a horse unquestionably comprises the examination of the urinary organs and of the urine. Without this many diseases may be overlooked.

Before actually examining the urine itself, the urinary organs and neighbouring structures must be examined as thoroughly as possible. In horses the sheath, in dogs the prepuce, in female animals the vagina and uterus, and in male animals the prostate and testicles, should all be passed in review.

In inflammatory changes of these parts, such as catarrhal and suppurative inflammation of the prepuce in the dog, catarrhal and suppurative inflammation of the vagina and uterus in the cow, and inflammation of the prostate in the dog, the urine is always mixed with a greater or less quantity of catarrhal and puriform discharge which influences the analysis in an important degree. The urine then contains a greater or less quantity of albumen, and, on microscopical examination, reveals the presence of cellular elements, such as pavement epithelium (which usually exhibits fatty or granular degeneration), cells from the vaginal and uterine mucous membrane, from the prepuce and sheath or from the prostate, as well as spermatozoa, numerous leucocytes, and large numbers of micro-organisms.

Should the results of examination exclude the above-mentioned diseases, the changes in the urine must be referred to some pathological change in one or other portion of the urinary apparatus, provided always that no constitutional or infectious disease exists which might lead to changes in the reaction or specific gravity, or in the albumen or sugar contents of the urine.

Physical examination of the urine comprises observation of the quantity, colour, transparency, consistence, reaction, specific gravity, and the presence or absence of sediment. Chemical examination is directed to the discovery of albumen, bile, colouring material, indican, blood, sugar, melanogenic substances, chlorides and phosphates, and also to the detection of certain drugs like salts of salicylic acid, antipyrin, iodine, bromine, morphine, sulphonal, turpentine, etc. This complete, the sediment should be subjected to microscopical examination.

The sediment is obtained either by deposition in a precipitating glass or by the use of a centrifugal separator. On account of the saving in time and the avoidance of fermentation, etc., the latter method is to be preferred.

The first portion of the examination should be made with a magnifying power of sixty to one hundred diameters, followed by the use of higher powers, up to six hundred diameters.

Crystals, some of which form a normal portion of the urine, are at once detected.

The urine of the horse usually exhibits diffuse cloudiness when passed. In the case of other herbivora this cloudiness is also seen after the lapse of a varying time, and is due to the presence of yellowish-brown, round, flattened, radially-striped crystals of calcium carbonate. These can be distinguished from similar spherical crystals like those of leucin, fat, etc., by adding a little acetic acid, when they dissolve with free disengagement of CO_2.

The crystals of calcium carbonate are mingled with little globular crystalline masses of tricalcium and trimagnesium phosphate.

Hippuric acid and calcium hippurate also form normal constituents of the urine of herbivora, and can be obtained by evaporation. The crystals are

recognised by their size, and by their assuming the form of four-sided rhombic prisms and needles. They are insoluble in hydrochloric acid, and can thus be distinguished from the crystals of triple phosphate, which they otherwise resemble.

In the acid urine of carnivora we find beautiful, highly refractive, and extremely small crystals of calcium oxalate, in the form of four-sided octahedra which, when seen from above, exhibit the outline of a commercial envelope.

The urine of herbivora may also contain calcium oxalate, though in somewhat larger crystals. This is more commonly the case in certain conditions like tetanus, in diseases of the digestive organs, in hæmoglobinæmia in the horse, in septicæmia, etc. The acid urine of carnivora on cooling often deposits crystals of uric acid and its salts, like the urates of magnesium, calcium, and sodium. Urate of ammonia, however, is not present, though it may be met with after the so-called alkaline fermentation of the urine.

The crystals of uric acid vary in size and form; their colour ranges from yellow to intense red. The commonest form is that of a rhombic tablet. They can be obtained in a very beautiful form by adding acetic acid to fowl dung (which consists principally of uric acid salts, such as the urates of calcium, magnesium, and sodium). The acid dissolves the uric salts, which after a short time deposit rhombic crystals of uric acid. Uric acid also takes the forms of whetstones, barrels, rosettes, spearheads, needles, etc.

After prolonged fever or starvation the urine of herbivora may also exhibit spear-headed and needle-shaped uric acid crystals in consequence of the urine assuming an acid reaction.

Uric acid and its salts may be detected by the murexide test. A small quantity of urine is mixed with a few drops of nitric acid in a porcelain bowl, and cautiously evaporated until only a yellowish-red dry residue remains. If after cooling this is treated with ammonia a fine purple-red colour is produced. Caustic potash gives a blue-violet tint.

This test enables uric acid and its salts readily to be distinguished from similar crystalline forms, such as those of calcium sulphate, which also occur in horses' urine when very acid in the form of columnar prisms and needles, and from the needle-shaped crystals of tyrosin, which, with crystals of leucin, may sometimes be found in very severe cases of metabolic disease, and usually assume a characteristic fascicular form.

The fine, large, strongly refractive rhombic prisms of the triple phosphate of ammonium and magnesium are not uncommon; their oblique end surfaces recall the shape of coffin lids.

These crystals are chiefly seen in cases of so-called "ammoniacal fermentation of the urine," a condition set up either in the pelvis of the kidney or in the urinary bladder, in cases of inflammation of the kidney or bladder, or after long exposure of urine to the air, in consequence of decomposition of the urea, with the formation of ammonia and CO_2 through the action of the micrococcus ureæ and bacterium ureæ. When the originally acid urine of carnivora is thus rendered alkaline, the substances soluble in acid solutions are deposited. These comprise the amorphous calcium phosphate, ammonium urate, crystals of which resemble thorn apples (datura stramonium), and the above-mentioned triple phosphate of ammonium and magnesium. The discovery of these three forms of crystals alone justifies the conclusion that the urine has undergone alkaline decomposition. Alkaline as well as acid urines may undergo this ammoniacal decomposition. In icteric diseases and in cases of hæmorrhagic nephritis and crystitis, yellow or reddish-brown, needle-shaped, spherical, or star-shaped crystals of bilirubin or hæmatoidin may occasionally be met with.

Fat may pass into the urine and be met with in the form of drops or needles, visible under the microscope. This occurs in cases of chyluria

accompanying eustrongylosis renalis in the horse, ox, and dog, and in fatty degeneration of any portion of the urinary system, as well as in cases of severe suppuration, malignant tumour-growth, cirrhosis of the liver, and poisoning with phosphorous or carbonic oxide.

Cystin, which Smith believes is due to putrefactive changes in the bowel, occurs in six-sided plates, and in carnivora may give rise to calculus formation.

Deposition of successive layers of the above-described crystals, and their cementation by mucus, leads to the formation of so-called urinary calculi. Depending on the nature of the constituent crystals, calculi are clinically grouped into those formed of carbonates, oxalates, phosphates, urates, cystin, and of mixtures of two or more of these.

The epithelium found in the sediment of urine is of special interest, for, whilst to some slight extent epithelium is shed and passed with the urine, even in healthy animals, the quantity becomes immensely greater whenever any inflammatory processes, whether of a catarrhal, purulent, hæmorrhagic, or diphtheritic nature, exist, and its character indicates the special point in the urinary apparatus from which it originated. Although in all these diseases, whether of the nature of catarrhal purulent urethritis, catarrhal purulent cystitis, purulent pyelo-nephritis, or simple nephritis, the urine always contains a greater or less amount of albumen, and varies in specific gravity and chemical reaction, these points are far from sufficient on which to base the diagnosis, and should be supplemented by microscopical examination of any existing epithelium, the recognition of its source, and the identification of any other organised material in the sediment obtained. The size of the single epithelial cells, apart from the size of the individual animal, depends on their points of origin. Those from the urinary passages, bladder, ureters, and mucous membrane of the pelvis of the kidneys are large; those, on the contrary, from the tubules of the kidney are small.

The form of the cells enables one to distinguish the flattened stratified epithelium from the mucous membrane of the urinary passages, bladder, etc., from the cylindrical epithelium of the urinary tubules. The former cells are of unequal breadth and usually provided with long processes. When local inflammation is acute they are frequently united, forming large bands. The latter, on the contrary, are slender, cylindrical in form, polygonal, and rounded.

Both varieties of epithelium contain nuclei. The nuclei of the epithelium covering the mucous membrane of the bladder are usually oval, those of the epithelium of the kidney rounded and relatively large. Microscopical examination, apart from other signs, also enables us to distinguish quite clearly between cystitis, urethritis, and pyelitis on the one hand, and nephritis on the other, simply from the size and form of the epithelium discovered.

It is not possible, however, exactly to differentiate between catarrhal cystitis and catarrhal urethritis. So far as prognosis and treatment are concerned, it is of little importance whether the condition be simple catarrhal cystitis, or be complicated with some disease of a catarrhal nature in the urethra. The diagnosis of nephritis and the differentiation between various forms of nephritis is greatly assisted by microscopical examination of the different casts.

With the exception of chalk casts, which may be found in the urine of healthy horses, all other kinds are pathological.

These casts vary in length, are usually parallel-sided, sometimes twisted, sometimes straight, and represent material exuded into the urinary tubules. They are produced during the course of nephritis in consequence of exudation occurring into the lumen of the urinary tubules and there coagulating.

In subacute and acute parenchymatous desquamative nephritis the cast

may either be entirely covered with unchanged epithelium from the urinary tubule (epithelial cast), or this enveloping epithelium may show signs of advanced degeneration, and the cast itself may either appear coarsely or finely granular (granular cast).

Should the nephritis follow a more chronic course (chronic interstitial nephritis), hyaline casts (which in consequence of their softness and yellowish-white tint are apt to be overlooked) and colloid waxy casts may be detected microscopically. The hæmorrhagic and purulent character of the nephritis is indicated by the presence of red and white blood corpuscles, and by the existence of casts composed of these cells.

In severe cases of hæmoglobinæmia in the horse (usually associated with parenchymatous nephritis), the epithelial and granular cylinders are accompanied by so-called hæmoglobin casts, which can readily be recognised in the dark, reddish-brown sediment on account of their golden-yellow or golden-brown colour.

It is only possible to differentiate exactly between hæmoglobinuria and hæmaturia by microscopical examination. In hæmoglobinuria formed elements of the blood are absent; in hæmaturia, on the contrary, they are present in large numbers. The red blood corpuscles seldom preserve their usual size and form, but are either shrivelled and resemble in outline minute thorn apples (datura stramonium), or are greatly swollen.

The discovery of red and white blood corpuscles, however, is not sufficient to indicate the seat of disease, which can only be arrived at by consideration of the other signs.

The presence of numerous red and scattered white blood corpuscles, together with large epithelial cells from the mucous membrane of the bladder (some exhibiting processes), and crystals of triple phosphate and acid urate of ammonium, indicates the existence of hæmorrhagic cystitis. Hæmorrhagic parenchymatous nephritis, on the other hand, is characterised by the presence of epithelium and cylinders from the kidneys, in addition to the numerous red and isolated white blood corpuscles.

The giant palisade worm (eustrongylus gigas), which affects various domestic animals (the horse, ox, and dog) has its seat in the kidney or pelvis of the kidney, and often produces hæmorrhagic nephritis. This form of eustrongylosis renalis is occasionally due to the presence of female parasites, and the brown-coloured, oval eggs of double outline may be detected on microscopical examination.

Furthermore, in botryomycosis of the kidney in horses, one may occasionally find in the urine masses of the parasite resembling blackberries in form.

In carcinoma of the kidney cancer cells may be found, and in advanced cases of carcinoma of the urinary bladder the microscopical appearances often confirm the diagnosis.

Tubercle bacilli are rarely found in the urine, though the discovery may be made in bi-lateral hæmatogenous tuberculosis of the kidneys of an advanced type. This condition is occasionally noted in oxen and dogs.

On the other hand, the bacillus pyelonephritidis bovis, which destroys the pelvis of the kidney and the parenchyma of that organ in the ox, is relatively common. Stained by Gram's method, it appears under high powers as a blue rod, 2 to 3 μ long and ·7 μ wide, slightly curved on itself, rounded at the ends, and usually collected in dense masses. Discovery of the typical bacillus, accompanied by characteristic epithelium from the parenchyma and pelvis of the kidney, crystals of triple phosphate of ammonia and magnesia, scattered red blood corpuscles and numerous leucocytes, justifies the diagnosis of bacillary pyelo-nephritis being made with confidence.

Microscopical examination of the urine therefore enables us exactly to diagnose an entire series of diseases which cannot be differentiated by clinical symptoms

alone, and in some instances to clear up what would otherwise be extremely obscure cases. On account of the frequency of disease of the urinary apparatus, and the injurious influence such disease exerts on the entire organism, it is desirable that the urine should be examined in all animals which have to be tested for general soundness and usefulness, and also in cases of disease where disturbance of other organs has been excluded. (*Berliner Tierärztl. Wochens.*, 22nd May, 1904, p. 398.)

A CASE OF BLACK–QUARTER IN A THREE–DAYS–OLD CALF.

By L. STREBEL, Junr.

OF thirty-three cattle on a certain farm in V——, twenty-eight had been protected against black-quarter in the spring of 1903, five being omitted because they were still too young. On the 5th November 1903, one of the uninoculated animals, a nine months old bull calf, died from black-quarter, as shown by *post-mortem* examination.

The prescribed disinfection was at once carried out. On the 7th November the four other young animals were inoculated for the first time.

On the evening of the 8th November, a cow in an entirely separate stable gave birth to a well-developed and apparently quite healthy calf. On the 11th November this calf died. Being called by the owner to make a *post-mortem* examination, Strebel discovered most clearly marked macroscopical lesions of black-quarter.

A bacteriological examination in the pathological laboratory at Freiburg proved the existence of black-quarter bacilli.

Strebel recounts the case because to his knowledge no case of black-quarter in so young a calf (only three days old) has for long been published. (*Schweizer—Archiv f. Tierheilk, XLVI. Band, 2 Heft, page* 86).

TWO CASES OF RECOVERY FROM EXPERIMENTAL RABIES IN THE DOG.

By MM. REMLINGER and MUSTAPHA EFFENDI.

IN no other disease, perhaps, is the prognosis less hopeful than in rabies.

In the laboratory it sometimes happens that a dog inoculated with the virus of rabies by trepanation escapes infection. Such an animal is regarded as having been protected by a previous attack, a view first suggested by M. Pasteur, but so far as the authors are aware the process of recovery from rabies has never been observed with absolute certainty, and has never yet been realised under laboratory conditions; hence the interest of the two following cases.

CASE I.—On the 7th November 1903, a full-grown, dark yellow, ownerless dog was injected in the jugular vein with 5 cc. of a milky emulsion of fixed virus previously passed through fine muslin. No special symptoms were noted until the 21st November, when the animal showed restlessness and considerable excitement. It wandered backwards and forwards in its cage, although during the previous days it had remained lying down. The appetite, previously good, had diminished.

On the 22nd November excitement was less, but the hind limbs showed signs of paresis, appetite was completely lost, the eyes appeared haggard, and and there was a little froth about the lips.

On the 23rd November the condition was aggravated. The animal lay continuously, and if forced to rise it rolled about and fell again immediately. Touching the animal or making any sudden noise caused convulsive attacks, which were particularly marked when dogs in the neighbourhood barked.

By the 24th November all excitement had disappeared. The hind limbs were completely paralysed, the animal lay on its outstretched fore limbs; it had not lost consciousness. If spoken to it moved its tail and slightly lifted its head. Dyspnœa.

On the 25th November the paralysis had extended to the muscles of the neck, the animal could no longer lift its head, which it rested on the ground, hind and front limbs completely paralysed, respiration feeble, consciousness lost; the animal appeared likely to live only a few hours.

26th November. Condition stationary; respiratory movements were so little marked that the animal sometimes appeared dead.

27th November. The observers were surprised to note that not only was the animal living, but it had sensibly improved. It had recovered consciousness, and when spoken to it moved its tail.

28th November. Continued improvement, the head could be lifted slightly; being unable to use its paws, the animal when called tried to approach by dragging itself along the floor on its arms. It could not eat, but tried to grasp fragments of bread placed within its reach.

29th November. Very marked improvement. The animal made several movements with its front and hind paws; when called it partly raised its body. It masticated and swallowed pieces of bread placed within its mouth.

1st December. Improvement continued. The dog used its paws better. It grasped fragments of bread by itself and ate them without assistance.

2nd December. When called for feeding it made an effort, rose on its legs, rolled about, fell down, rose again, rolled about afresh, and repeated this a certain number of times. Appetite increased.

3rd December. The animal was found standing up in its cage, propped against the bars to avoid falling. If forced to leave its position it rolled about and fell down, but rose again with much less difficulty than on the previous day.

On the following day the improvement steadily continued. The hind limbs still showed paresis, and the gait remained staggering for some time, but by the 10th or 15th December the animal could be regarded as completely cured. The only signs of disease were poor condition and perhaps some loss of visual acuteness.

Given the above-described symptoms, and remembering that they occurred some fourteen days after an intravenous inoculation of fixed virus, it seems impossible to attribute them to anything but an attack of rabies. Nevertheless, it proved impossible to obtain froth from the mouth during the attack, to inoculate an animal sensitive to the disease. To confirm their diagnosis the authors therefore sought to discover if the serum had assumed anti-rabic powers, and if the animal itself had acquired immunity.

On 14th December the dog was bled. On the 16th the serum was emulsified with fixed virus. Virus and serum were left in contact for twenty-four hours, after which the serum was separated. The virus was diluted with sterilised water, and two rabbits were inoculated under the dura mater. One of these died of paralytic rabies on the 31st December, the other on the 1st January. In these animals therefore death had been postponed for a period of four and five days respectively.

On the 5th January the dog was trephined and inoculated with a large dose

of fixed virus. No morbid symptoms followed. Two months later it was still alive and perfectly well.

As a control the authors inoculated, in the anterior chamber of the eye, a dog which one month previously had received by the jugular vein 10 cc. of rabic emulsion and had shown no morbid symptoms whatever as a consequence. This animal died of rabies three weeks later. It follows that the fact of not having shown rabies after trephining should not be attributed to the inoculation of virus into the jugular, but, on the contrary, to the action of the disease process which followed. The rabic character of the latter is thus proved.

The authors describe a second case in which an ownerless dog received 8 cc. of a milky emulsion of fixed virus in the jugular vein. The first symptoms, viz., loss of appetite and slight paresis of the hind limbs, were noted on the 10th December, twelve days after inoculation. The subsequent course of the disease was very similar to that in the case already detailed. The animal appeared completely to recover, except in so far as its bodily condition and vision were concerned.

26th December. The animal was bled.

28th December. The serum was mixed with fixed virus.

29th December. A rabbit was inoculated under the dura mater with the virus which had been freed from the serum and diluted with sterilised water. This rabbit was attacked on the 10th January, thirteen days after inoculation, and died on the 13th January, that is, fifteen days after inoculation. In this case death was postponed for five days longer than in the case of the control animal.

On the 5th January the dog was trephined and inoculated with a large dose of fixed virus. No morbid symptoms. Two months later it was still alive and in excellent health. The control animal kept under similar conditions had contracted rabies.

The authors noted that in the second dog the disease was much less marked than in the first. The two cases therefore represent a gradual transition between those cases where inoculation of rabic virus into the jugular vein produces no effect (four times in ten in this series of experiments) and those on the contrary where it produces death after an attack of classic rabies (four times in ten as before). A modified attack of rabies often confers immunity against so severe a test as inoculation under the dura mater, the serum of a dog immunised in this way possessing anti-rabic properties. These facts suggest the possibility of vaccinating dogs against rabies by way of the jugular vein as practised by Krasmitski.

The authors' observations, however, only partially confirm the experiments of that writer, who regards intravenous injections of rabic virus as harmless, provided the emulsion be filtered, diluted, and injected slowly. The difference in the results obtained is probably connected with the degree of concentration of the emulsion.

The foregoing facts should direct attention to a second point. If experimental rabies is susceptible of cure, the same is undoubtedly true of clinical rabies. In that case a person bitten by an affected animal cannot certainly feel safe, as has hitherto been supposed, even although the animal remain alive eight to ten days after the accident. The survival of the dog is not an absolute criterion. The dog may fatally inoculate a human being with rabies whilst itself surviving. A very searching veterinary examination should therefore be made, and in all doubtful cases the Pasteur treatment should be carried out. (*Annales de l'Institut Pasteur*, 25*th* April 1904, *page* 241).

TUBERCULOSIS OF THE BRAIN IN A COW.

By M. BERGEON.

BERGEON was called on to examine an eight years old cow whose appetite had for some time been capricious. The animal walked unsteadily, had difficulty in rising, and showed periods of coma succeeded by phases of excitement. Despite treatment (by a quack), it had steadily become worse. At the time of examination it was in fair bodily condition.

When eating hay the cow commenced by masticating slowly, dropped the mass of food into the manger, picked it up again, remasticated it, and finally swallowed it with difficulty. Liquids and sloppy foods were taken in a somewhat peculiar way. After a preliminary inspiration, the cow suddenly plunged her muzzle to the bottom of the pail, keeping it there till short of breath, when she withdrew it, afterwards slowly sucking up the contents. The head was depressed and inclined towards the right; the neck was turned in the same direction. The eyes had lost expression and appeared vacant.

The animal had difficulty in rising and rested for a moment on her knees. In walking she hesitated and had difficulty in co-ordinating the movements of the limbs : the head was carried low, turned towards the left, and there was some tendency to walk in a circle in that direction. The horns and frontal region were of normal temperature, the pulse slow and irregular, the rectal temperature 38° C.

Bergeon suspected some chronic cerebral lesion, and to dispose of the question of tuberculosis injected tuberculin. A well marked reaction followed, the temperature rising 2¼ degrees C. The cow was therefore slaughtered.

On *post-mortem* examination numerous tuberculous granulations were found in the lungs, and on the pleura and pericardium. The pectoral glands were enlarged and indurated, and contained typical caseo-cretaceous pus.

The meninges of the brain were thickened and congested, but the latter sign was quite secondary, the cow having been slaughtered with a pole axe. The same was true of the congestion over the mass of the brain ; but, on incising the latter, Bergeon found in the depths, opposite the right frontal lobe, a yellowish new growth as large as a walnut, with caseous contents, which he regarded as the cause of the disturbance shown and as evidently tuberculous. (*Revue Vétérinaire*, 1st May 1904, p. 321).

ANEURISM OF THE RIGHT INTERNAL THORACIC ARTERY IN THE OX.

By J. B. PIOT BEY.

IN describing a case of aneurism on the left coronary artery in 1896, the author mentioned the extreme rarity of these lesions in oxen before adult age, and pointed out that one of the principal causes of this rarity was the fact that most animals were slaughtered before arriving at the period in question. The new case of aneurism described below has led him to modify his conclusions.

Despite careful perusal of most of the veterinary journals and the ancient and modern classical veterinary writers, he has only been able to find a few cases in the ox, whilst in the horse cases are to be found by hundreds and in the human species by thousands.

There appears no question as to the rarity of aneurism in bovine animals during their ordinary period of existence, or until they are slaughtered at the

age of seven to eight years, and the author even goes further and says that aneurism is excessively rare even in oxen of extreme old age.

Opportunities for studying this question are probably more abundant in Egypt than in any other country, for working oxen are there kept until completely worn out. The author claims to have had under observation more than thirty thousand working oxen varying in age between five and twenty-five years, until they became unfitted for work and often until they were slaughtered, and yet he has only seen two cases of aneurism, one of which is described hereafter.

His brother who, for the past fourteen years has had charge of the abattoir at Alexandria, in which from twenty to twenty-two thousand oxen, the majority of advanced age, are slaughtered yearly, has never seen aneurismal dilatations.

Below is a short description of the second case of aneurism in the ox which Piot Bey has seen amongst the animals belonging to the Administration of Domains.

On the 21st April last he was called to examine an ox which had died suddenly. The skin had been removed and the abdomen was extremely distended. The ox was eleven years of age, had been resting for a fortnight, and, having been freely fed, was found one morning lying on its side with its neck and head in a state of extreme extension and its limbs moving convulsively. The animal died before it could even be bled.

On opening the abdomen all the organs appeared depleted of blood, without there being any other apparent alteration. This suggested internal hæmorrhage, a view confirmed immediately afterwards on withdrawing the abdominal viscera and finding that the right half of the diaphragm was strongly pressed towards the abdominal cavity by a soft mass, the dark colour of which was faintly visible through the substance of the diaphragm.

The diaphragm having been divided close to its insertion, an enormous clot, weighing quite twenty-five pounds, escaped in a piece together with a little reddish serosity, both coming exclusively from the right pleural cavity. The lungs and heart were then carefully removed from the thoracic cavity, and the seat of the rupture was sought in the heart and large vessels. The search proved fruitless. In the thoracic cavity, however, the point whence the hemorrhage had occurred was found near the anterior third of the sternum, concealed within a mass of fatty tissue from which an enlarged lymphatic gland projected prominently.

The right internal thoracic artery presented an aneurism, oviform rather than fusiform in shape, about five inches long, and two and a half inches in diameter.

The dilatation commenced suddenly at the point where the artery turns backwards after leaving the first rib, and terminated slightly in front of the point where the vessel passes under the triangularis sterni muscle.

The rupture was represented by a linear orifice about half to three-quarters of an inch in length on the supero-internal surface of the aneurism, towards its posterior third, and in a direction oblique to its long axis.

On longitudinal incision, the walls of the dilated part were found to be from two to three millimetres in average thickness, that is, very much greater than that of the normal wall of the internal thoracic artery. They only collapsed slightly and still appeared to preserve a certain degree of elasticity.

The aneurismal pocket was simple, and partially filled with recent clot, but showed no trace of old existing clot. The internal tunic showed large thin atheromatous patches marked by several helicoidal depressions, the most posterior of which was the seat of the rupture.

On the right side of this internal tunic was a kind of fungating patch, of a fine dark red velvety appearance, irregularly polygonal in form, two to three

millimetres in thickness, with thickened margins, and soft and friable in texture. To the naked eye this resembled a collection of small hæmorrhagic patches, and reminded one of a rich culture of bacilli on a solid medium.

After hardening the specimen in alcohol for twenty-four hours and preserving it in 10 per cent formalin, the patch became stiff, dry, and fragile like chocolate. (*Recueil de Méd. Vét.*, 30th May 1904, p. 335).

ROYAL COMMISSION ON TUBERCULOSIS.

INTERIM REPORT.

THE Royal Commission which was appointed on 31st August 1901, to inquire into the relations of Human and Animal Tuberculosis has issued the following interim report, which is signed by all the Commissioners, namely, Sir Michael Foster, M.P. (chairman), Professor G. S. Woodhead, Professor Sidney Martin, Professor M'Fadyean, and Professor R. W. Boyce :—

To the King's Most Excellent Majesty.

May it please your Majesty,

We, your Majesty's Commissioners, appointed to enquire and report with respect to tuberculosis :—

1. Whether the disease in animals and man is one and the same;
2. Whether animals and man can be reciprocally infected with it;
3. Under what conditions, if at all, the transmission of the disease from animals to man takes place, and what are the circumstances favourable or unfavourable to such transmission ;

humbly submit this report on the progress which we have made in the enquiry.

The greater part of the above reference is directed to the view which had been expressed that the bacillus which gives rise to tuberculosis in the bovine animal is specifically distinct from the bacillus which gives rise to tuberculosis in the human being, and that therefore the presence of the bovine bacillus in the milk or flesh of the cow, consumed as food by man, is not to be regarded as a cause of tuberculosis in the latter. To this point we first turned our attention.

After duly considering the matter, we came to the conclusion that it would be desirable not to begin the inquiry by taking evidence—that is to say, by collecting the opinions of others (though this might be desirable at a later stage), but to attack the problem laid before us by conducting experimental investigations of our own.

The first line of enquiry upon which we entered may be stated as follows :—

What are the effects produced by introducing into the body of the bovine animal (calf, heifer, cow), either through the alimentary canal as food, or directly into the tissues by subcutaneous or other injection, tuberculous material of human origin — *i.e.*, material containing living tubercle bacilli obtained from various cases of tuberculous disease in human beings, and how far do these effects resemble or differ from the effects produced by introducing into the bovine animal, under conditions as similar as possible, tuberculous material of bovine origin—*i.e.*, material containing living tubercle bacilli obtained from cases of tuberculous disease in the cow, calf, or ox ?

We have up to the present made use in the above inquiry of more than 20 different "strains" of tuberculous material of human origin—that is to say, of material taken from more than 20 cases of tuberculous disease in human beings, including sputum from phthisical patients and the diseased parts of the lungs in pulmonary tuberculosis, mesenteric glands in primary abdominal tuberculosis, tuberculous bronchial and cervical glands, and tuberculous

joints. We have compared the effects produced by these with the effects produced by several different strains of tuberculous material of bovine origin.

In the case of seven of the above strains of human origin, the introduction of the human tuberculous material into cattle gave rise at once to acute tuberculosis, with the development of widespread disease in various organs of the body, such as the lungs, spleen, liver, lymphatic glands, etc In some instances the disease was of remarkable severity.

In the case of the remaining strains, the bovine animal into which the tuberculous material was first introduced was affected to a less extent. The tuberculous disease was either limited to the spot where the material was introduced (this occurred, however, in two instances only, and these at the very beginning of our enquiry), or spread to a variable extent from the seat of inoculation along the lymphatic glands, with, at most, the appearance of a very small amount of tubercle in such organs as the lungs and spleen. Yet tuberculous material taken from the bovine animal thus affected, and introduced successively into other bovine animals, or into guinea-pigs from which bovine animals were subsequently inoculated, has, up to the present, in the case of five remaining strains, ultimately given rise in the bovine animal to general tuberculosis of an intense character; and we are still carrying out observations in this direction.

We have very carefully compared the disease thus set up in the bovine animal by material of human origin with that set up in the bovine animal by material of bovine origin, and so far we have found the one, both in its broad general features and in its finer histological details, to be identical with the other. We have so far failed to discover any character by which we could distinguish the one from the other; and our records contain accounts of the *post-mortem* examinations of bovine animals infected with tuberculous material of human origin which might be used as typical descriptions of ordinary bovine tuberculosis.

The results which we have thus obtained are so striking that we have felt it our duty to make them known without further delay in the present interim report.

We defer to a further report all narration of the details of our experiments (and we may say that up to the present time we have made use of more than two hundred bovine animals), as well as all discussions, including those dealing with the influence of dose and of individual as well as racial susceptibility, with questions of the specific virulence of the different strains of bacilli, with the relative activity of cultures of bacilli and of emulsions of tuberculous organs and tissues, and with other points. In that report we shall deal fully with all these matters, as well as with the question why our results differ from those of some other observers.

Meanwhile we have thought it our duty to make this short interim report, for the reason that the result at which we have arrived —namely, that tubercle of human origin can give rise in the bovine animal to tuberculosis identical with ordinary bovine tuberculosis—seems to us to show quite clearly that it would be most unwise to frame or modify legislative measures in accordance with the view that human and bovine tubercle bacilli are specifically different from each other, and that the disease caused by the one is a wholly different thing from the disease caused by the other.

In conclusion, we desire to express in the strongest terms our appreciation of the most generous assistance given to the Commission by Sir James Blyth, who has placed unreservedly at our disposal his farm buildings and other accommodation at Stansted. By his action not only has the nation been saved a very large necessary expenditure, but we have been able by the help of the admirable arrangements made for us to carry out our investigations in a

manner which would have been impossible had the accommodation and equipment for our enquiry been provided entirely at the public cost.

And we wish also to thank our secretary, Dr E. J. Steegmann, and our observers, Drs Louis Cobbett, A. Stanley Griffith, Eastwood, and Hutchens, as well as the rest of our staff, for the able services which they have untiringly rendered to us.

PROFESSOR KOCH'S FOURTH REPORT ON AFRICAN COAST FEVER.[1]

SINCE the publication of my last report, the method of protective inoculation therein recommended has been applied on a large scale to European and native owned cattle, in order to ascertain how it was likely to answer in actual practice. These field experiments began in October 1903, and are still going on, and the result of the work done is tabulated on the accompanying returns, to which I append the following commentary.

List " A " shows the work done in the Victoria district. Operations which commenced here on 24th October were initiated by Chief Veterinary Surgeon Gray, under my personal supervision, and subsequently kept up by Messrs Readman, Hill, and Southey, and to the expeditious manner in which the work can be carried out I can bear witness.

To obviate any difficulty which might be experienced in obtaining animals in Victoria suitable for inoculating from, five salted cattle from Hillside Camp, in whose blood the ring forms of the parasite were present, were sent on to await our arrival. These animals were subsequently supplemented by other suitable animals purchased locally.

A large proportion of the cattle in this district being owned by natives, before we left Buluwayo the Acting Native Commissioner, Mr Forrestall, sounded native stock-owners as to their willingness to have their cattle inoculated. The majority expressed themselves as anxious to have their animals treated, and as European farmers were equally unanimous in their desire to give the method a trial, little time was lost in making a start, and thanks to the interest taken in the work by Mr Forrestall, who accompanied us everywhere and acted as interpreter, we had no difficulty in making rapid progress, the presence of the Native Commissioner having a reassuring effect on the natives, who were at first inclined to look doubtfully on a method of inoculation in which blood was used.

The usual daily tale of animals inoculated in the first round at Victoria ranged between 200 and 400, depending upon the distance traversed between the various kraals. On several occasions Mr Gray inoculated over 500 in a day, but such days were exceptionally heavy; 300 head a day may be taken as a very fair average and that number could only be got through comfortably when a considerable number of natives were present to catch and hold the animals so that no time was lost in going from one animal to another.

Shortly before the work began the rains commenced, and with the commencement of the wet weather the disease everywhere showed a tendency to revive. To such an extent was this the case that instead of having to inoculate, as we anticipated, 1000 head of cattle in order to include all animals in infected herds and their vicinity we found that over 4000 had to be dealt with. In almost every direction we met cases of the disease, and microscopic examination of blood smears from sick and dead animals at Mangwendi's and Matchokoto's

kraals near the township showed that the type of the infection which prevailed was a severe one.

The history of the course of the disease in Victoria is much the same as in other districts.   It was originally introduced a year ago by transport cattle from the north, first appearing on the Victoria-Enkeldoorn road, then the commonage was infected in spite of precautions taken to preserve its integrity by prohibiting the outspanning of transport cattle within the boundaries and enforcing compulsory dipping of all animals entering the township.

Once the disease became established on the commonage it made a clean sweep of most of the cattle grazing there, and native stock owners residing some distance from town, who had cattle whose milk was sold in town, running at the kraals on the edge of the commonage, took fright and moved their cattle from the vicinity of the infected area, in many instances carrying the disease with them to their kraals, so when we arrived at Victoria we found the commonage for the most part denuded of cattle and beyond it a thickly stocked area where isolated outbreaks had occurred, surrounded in most cases by herds of cattle amongst which there had been no outbreaks, although some of these were probably already infected and all were in imminent danger of becoming so by grazing in the vicinity of infected herds.   The apparently clean herds were in such a precarious position that the natives willingly agreed to their being inoculated, and they were, therefore, included in the sphere of operations and helped to swell the sum total of animals treated.

Such herds, amongst which no cases of African Coast Fever had appeared, are classified on the return under two headings.   Those in which the animals remained healthy during the inoculation period are characterised as " clean herds."   Those in which isolated cases of the disease occurred shortly after inoculation began are shown as " doubtfully clean herds."

That the mortality amongst " doubtfully clean herds " is due not to inoculation but to veldt infection is readily apparent from a consideration of the positions of the herds included under this category in return " B," the Chibi return—for all the herds shown there under this heading are herds whose grazing field overlaps that of infected herds, therefore these herds are included in the general return (Sheet D) amongst infected herds.

The third group on the returns headed "infected herds" requires no explanation.   It comprises herds infected at the time inoculation was begun.

Group No. 4 is shown as " combined infected and clean herds."   These are herds in which the cattle owned by one individual or kraal were broken up into separate batches, some of which were clean, and some infected.   There are two lots of cattle of this description shown in the Buluwayo return (sheet C) and one in the Victoria return (sheet A), and they are interesting and instructive in so far as inoculations were conducted simultaneously in both clean and infected herds, the same sample of blood being used for each.

Reference to List " A " shows that at Victoria thirty clean herds, containing 1701 head of cattle, received up to date a maximum number of seven inoculations.

The addition to these of the clean herd of Chikangangs brings the total number of clean inoculated cattle in this district up to 1809, amongst which the mortality up to the time of writing is nil.

To the number of cattle in infected herds shown as such in List " A " there must be added the cattle of " doubtfully clean herds " and those of Chikanganga's infected herd.   Summed up, the animals in these herds amount in all to 710 head, amongst which there have been fifty-two deaths.   In most infected herds so far only isolated cases of disease have been recorded, although in a few instances (Bhututu, Mangwendi, and Matchokoto) there has been a relatively high death rate, a circumstance to which I will refer later on.

Sheet " B " showing the work done in the Chibi district (35 miles from

Victoria), call for no special comment. This district was infected from Victoria, and the disease having appeared there more recently inoculation was begun at a somewhat later date, so that six inoculations is the maximum number which has been made in this district. Otherwise the conditions are similar to those obtaining in Victoria. Here twenty-four clean herds, comprising 1228 cattle, are being inoculated without any mortality resulting so far. Besides these, ten infected herds of 656 are also under treatment, amongst which there have been sixteen deaths.

Sheet "C" shows work done in and around Buluwayo. The disease appeared here about twenty months ago, and while some parts of the district are highly infected, there are still a number of places in the vicinity of the town which are clean. Our opportunities of dealing with clean herds in this district were limited to two, one a herd belonging to Colonel Napier and the other a herd belonging to Mr Heberden, which must be considered clean although eight deaths from African Coast Fever had occurred in it a long time before inoculation commenced. The latter herd Mr Heberden succeeded in freeing from the disease by moving it to fresh pasture after each outbreak. Such a line of procedure seems to me of great value, and recourse should be had to it whenever local conditions permit of movement without endangering the safety of neighbouring herds. Mr Heberden's herd has been inoculated thirteen times and remains healthy.

I am sorry to say that in several instances cattle owners who began inoculating suspended operations after a few injections because no immediate benefit was observable, although expectations that such would be forthcoming were scarcely justifiable, as I pointed out in my third report that good results need not be looked for when inoculation is applied to herds in which the disease has gained a thorough foothold.

As evidence of the futility of inoculation, public reference has frequently been made to a herd belonging to Mr Fleming, a farmer, resident in the vicinity of Bulawayo, but that such a case does not demonstrate the inefficacy of the method can be clearly shown. The herd in question consisted of nine animals, the survivors of about thirty which were alive a year ago. These nine animals were first inoculated on 15th October 1903. Four injections were made, and then the owner refrained from proceeding further with the experiment, because two or three weeks after the date of the first injection two animals died of African coast fever. How many more of the inoculated animals have since died I do not know, but last week blood smears taken from two calves belonging to the same individual were examined at the laboratory, and were found to give evidence of severe infection, and these animals in all probability have since died.

As a matter of fact this herd should not appear in the return at all because inoculation operations were discontinued, but I include it lest the public should think that a record of this case was deliberately omitted because its outcome had not been satisfactory.

In this district, as at Victoria, we find instances (Erasmus and Fingo location) in which owners have clean and infected lots of cattle which were inoculated simultaneously with the same blood without detriment to the healthy herds, which remain in perfect health.

Altogether 378 animals in clean herds have been inoculated in and around Bulawayo without loss. Of infected herds 312 head have been dealt with, of which 106 have died.

The sum total of the results of the experiments in the Bulawayo, Victoria, and Chibi districts are shown in return "D," and the conclusions to be drawn from our work so far, are :—

First.—Inoculation is devoid of danger, therefore cattle may be inoculated without any risk being incurred.

M

Second.—In infected herds a mortality of about 10 per cent. has occurred during the period of inoculation.

If we wish to arrive at some estimate, from a consideration of these figures, of the number of animals that have died because the recommended method has failed to protect them, the following facts must be borne in mind :—

First.—The method is protective not curative. For this reason all deaths occurring within twenty-eight days of the first inoculation must be excluded, as animals dying within that period were probably infected before treatment began, allowing for an incubative period of fourteen days in such cases, and a similar period for the duration of the disease.

Second.—Immunity does not begin immediately after the first inoculation. The time required for its establishment is greater than I first supposed. The experiments made at Hillside Camp show that animals turned into a highly infected pasture will not resist infection after eight weeks treatment, and I think that the immunising effect of the process is only beginning at the end of that time, and requires at least four or five months before it is completed, therefore any death occurring within three months of the date of the first inoculation cannot be attributed to the inefficacy of the process, because such deaths, in all probability, have occurred amongst animals which have become infected before they have been eight weeks under treatment.

Looked at from this standpoint, the results obtained in infected herds assume quite a different aspect, as consideration of a detailed account of the few cases in which we were able to obtain precise information tends to show.

The cases to which I allude are those of Bhututu, Mangwendi, and Matchokoto in the Victoria district, and that of the Fingo location in the neighbourhood of Bulawayo.

I will take first that of Bhututu. The herd at Bhututu's kraal originally consisted of about 100 head of cattle. In the course of four or five months seventy animals died, and the surviving twenty-six were inoculated for the first time on 24th October. Of this severely infected herd three animals died during the next three months, that is, within the period which precedes the development of immunity.

Next in order is Mangwendi's herd. This herd consisted originally of seventy-three animals grazing in the immediate vicinity of Bhututu's kraal. Seventeen animals died before inoculation began, and at the time of the first inoculation we found one animal dead and two sick, both highly infected. The two which were sick died during the week and are included in the mortality return. Within the first two months of inoculation thirteen died, including the two aforementioned. During the month of January (the third month after the commencement of inoculation) twelve more dropped off.

In Matchokoto's herd, which originally contained about fifty, sixteen only were alive ten days prior to inoculation, and on the day of inoculation seven were left (four adults and three calves), one of which was very sick and died the following day. Since then four more have died, leaving two survivors out of a herd of fifty. No good result was anticipated from the inoculation of this herd, and it might very well have been left alone, but it was considered advisable to inoculate as the natives were desirous that their cattle should be treated.

At the Fingo location near Bulawayo cattle to the number of about 650 were running when the disease first appeared amongst a lot of 437. Of this infected herd, 350 died previous to 21st October, the date upon which inoculation began. During the month of October eighteen died. How many of these died prior to inoculation I could not ascertain, therefore all are shown in the mortality return. In the month of November there were fourteen deaths, in December four, and in January thirteen. Of all these we really ought to

reckon that only those dying in the last week of January died in spite of inoculation.

I think a careful consideration of the detailed cases of Bhututu, Mangwendi, Matchokoto and the Fingo location makes it clear that not more than a small percentage of the total mortality of 10 per cent. shown as having occurred in infected herds can be said to have happened because the animals had not been benefited by the recommended treatment.

I do not contend, however, that the results of our field experiments prove that the method so far has afforded a high degree of protection of the stock which have been subjected thereto. Sufficient time has not elapsed for this to be apparent.

Our work only shows that the establishment of artificial immunity is a much more gradual process than I first hoped it would be.

With regard to the suitability of animals for inoculating from, we find comparatively little difference in the effect produced by the blood of various animals with which we carried out our inoculation, but more marked benefit appeared to be derived by the use of blood drawn from animals which had recovered from a real attack. Our experiments also show that 5 cc. of defibrinated blood is a sufficient quantity for each injection, and a regular system of fortnightly inoculations I find is best. By the preliminary weekly injections recommended in my former report, I hoped to hasten the establishment of immunity, but I am now convinced that it is impossible to force the process.

I feel certain that in the course of the next few months proofs of increased immunity in inoculated herds will be forthcoming, therefore I strongly recommend the continuance of the field experiments already begun, modifying the original process by using 5 cc. of recovered blood instead of 10, and allowing an interval of a fortnight between each injection.

It is a matter for regret that immunity is established so slowly, but the method recommended is satisfactory in this respect:—that once animals are thoroughly immunised no further treatment will be necessary, for they will be permanently immune, and their progeny after contracting the disease in a mild form will also resist further infection.

While I have called attention in previous reports to the fact that this disease has only been found in Rhodesia, in the Transvaal, and on the African coast, it is of great interest to note that Dschunkowsky, in No. 4 of the *Centralblaat fur Bakteriologie* lately published, describes a disease pre valent in the Russian Transcaucasus which is very probably identical with African Coast fever, judging from the train of symptoms described as occurring in infected animals and from the drawings given of the specific parasite. I think it is not improbable that a more extended search will show that the disease is much more widely distributed than we at first supposed.

Conclusions.

I will close these reports on African Coast fever by stating briefly the conclusions which may be drawn from a consideration of our experiments and observations so far as these are of interest in enabling us to understand the nature of the disease and so far as they suggest what steps should be taken to combat its spread.

First.—African Coast fever is a cattle disease caused by a characteristic blood parasite, and cases of the disease can be readily identified by a demonstration of the specific organism.

Second.—It is different from Texas fever or so-called redwater, which was introduced into and spread widely over South Africa before the advent of African Coast fever.

Third.—African Coast fever may be combined with redwater, and hæmoglobinuria (blood-coloured urine) is only observed in such cases. It was the occurrence of cases of mixed infection of this class which rendered the identification of the disease so difficult when it first came into this country.

Fourth.—The disease did not originate in Rhodesia but was introduced from the Coast.

Fifth.—It is not transferable directly, and sick animals can be stabled with healthy ones without communicating the disease.

Sixth.—The disease can only be spread by ticks. In this respect it resembles Texas fever.

Seventh.—The mortality from African Coast fever is very high, the loss in an infected herd commonly amounting to 90 per cent.

Eighth.—Animals which survive are protected against second attacks, and so long as they remain on infected veldt the immunity continues. That this is the case, and that the progeny of such animals are protected to a certain degree and become immunised by mild attacks of the disease during infancy, a consideration of the Coast fever areas on the East Coast of Africa proves.

Ninth.—In the manner referred to in paragraph 8 a really immune stock will be evolved in any country in which the disease appears.

Tenth.—Immune animals are not free from parasites.

There are always a small number of ring-shaped or ovoid parasites in the blood of such animals.

Eleventh.—By the transfer of parasites from salted to healthy animals by ticks the disease can be produced in a virulent form. This fact explains the mysterious infection of healthy stock by recovered and apparently healthy animals if herded together on the same veldt. The introduction of susceptible animals into a pasture over which salted animals have grazed and dropped ticks may suffice to infect them although they never come into actual contact with the immune herd.

Twelfth.—It is impossible to produce the disease by single injections of blood drawn from a sick animal and containing the parasites, although this can be readily done in Texas fever.

Thirteenth.—Repeated injections into healthy animals of blood containing parasites at intervals of fourteen days produce a very mild infection, characterised by a slight rise of temperature and the appearance of a few parasites in the blood. By such mild attacks animals so treated become immune after four or five months.

Fourteenth.—For immunising purposes the blood of a recovered animal can be used if the parasites are present, but care should be taken to see that such blood is free from the parasites of other South African diseases, such as trypanosomata and spirilla.

Fifteenth.—Inoculation *per se* can do no harm if ordinary care is taken to conduct the operation in a cleanly manner.

Sixteenth.—A clean herd can be protected by careful isolation from suspects, particularly from such animals as have had the disease and recovered from it. For this purpose fencing is beneficial.

Seventeenth.—The destruction of ticks by spraying or dipping is advantageous and helps to check the spread of the disease, particularly when combined with fencing. The discovery of a certain and safe method of destroying all ticks, which so far we have not at our command, would arrest any further spread of the disease.

Eighteenth.—After the disease has appeared in a herd movement of such a herd to clean veldt from time to time, particularly after outbreaks, is beneficial, and will sometimes suffice to free it from infection if the conditions are favourable.

Nineteenth.—Clean veldt is only such veldt upon which neither sick nor recovered animals have grazed for at least twelve months.

Twentieth.—Such expedients as fencing, dipping, spraying, and moving animals have only a temporary value, as sooner or later the disease will extend to and involve all herds in the vicinity of an infected centre, as Texas fever did; therefore such precautions should be supplemented by inoculation with recovered blood whenever disease appears in the vicinity.

Twenty-first.—The artificial establishment of immunity takes from four to five months. It is devoid of risk, inexpensive, and when animals are fully immunised no further treatment is necessary. Until this stage is reached animals should be protected as far as possible by spraying, fencing, and periodical movement.

	Number of Cattle Inoculated.	Number of Deaths.
Clean Herds—		
Victoria District	1509	—
Chibi District .	1228	---
Bulawayo District	378	—
	3115	—
Infected Herds—		
Victoria District	720	52
Chibi District .	656	16
Bulawayo District	312	106
	1688	174

REPORTS ON HORSE–SICKNESS BY HERR PRO-FESSOR DR. ROBERT KOCH.

FIRST REPORT.[1]

It is a fact well known to South African stockowners that horses which have recovered from horse-sickness are unlikely to sicken again from the disease, or if they do they generally contract it in a mild form. Such recovered animals are said to be "salted," a popular term indicating the acquirement of immunity under natural conditions. The knowledge of this fact—that salted animals are highly resistant to further infection, has led investigators for many years past to attempt to protect susceptible animals by producing attacks of horse-sickness by artificial means, in the hope that such artificial attacks would be less frequently fatal or more amenable to treatment than those occurring in animals naturally infected. Some experiments have succeeded, but so far the number of animals immunised in this manner has been small, and the attendant mortality has been so large that such methods have hitherto been regarded as impracticable, being too expensive and uncertain.

Many recorded experiments have been conducted on lines suggested by consideration of the good results obtained by the employment of serum in the treatment of rinderpest, and in some cases a serum for horse-sickness was obtained by the fortification of immune animals which possessed certain pro-tective powers, but, unfortunately, the severe and frequently fatal hæmo-globinuria following its use, due to its hæmolytic properties, debarred investi-gators from carrying on further experiments in this direction.

Science, however, is continually advancing. New methods of artificial immunisation are being discovered and old ones improved upon, and on this

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[1] This Report is dated 7th February, 1904.

account I considered it advisable to attempt to improve the serum method of immunising against horse-sickness, as this line of work seemed to be more promising than any other, and because I hoped that new methods might give better results.   For this purpose several salted horses were obtained, whose immunity I intended to heighten artificially, material for fortification being obtained in the first case from a horse suffering from horse-sickness, which Col. Bodle kindly sent to Hillside Camp.   This animal died on the road, the *post-mortem* revealing all the ordinary characterists of horse-sickness.   Blood taken from it was injected into several salted horses and one unsalted animal. The latter subsequently developed the disease and died, its blood being used to fortify the salted animals which remained perfectly healthy, and were therefore judged to be immune and suitable for fortification.   Subsequently these animals received big doses of virulent horse-sickness blood subcutaneously and intravenously at intervals of three or four weeks.   The maximum dose injected was 2,000 cc., which was sustained without any ill effect.   After a certain number of injections one fortified horse was bled, and its serum tested to determine whether it possessed any protective properties.   Experiment proved that it did have such properties, but only to a very slight degree.   In artificially infected animals injections of this serum merely delayed the incubative period and prolonged the duration of the disease.   I therefore continued the fortifying process for some months before again testing the serum.   At the second test it was found to be highly protective, and to ascertain its strength five horses were injected subcutaneously with various doses of serum, receiving the following day a uniform dose of virulent blood which had been previously tested and which had invariably proved fatal to susceptible animals.   One of the five horses inoculated—a young Australian animal four years old— developed the disease, recovered from it, and afterwards received successive injections of virulent blood without any harm resulting.

The other four inoculated animals remained perfectly healthy, and in order to determine whether this was due to natural immunity or to the protective power of the serum, sixteen days were allowed to elapse, and then three of them (two old animals and a young one) were again inoculated with virulent blood only, the dose being the same as that given in the first instance.

All three inoculated animals developed horse-sickness and died after the usual incubation period.

This experiment proves, first, that the horses used were susceptible.   Second, that the serum enabled them to resist inoculation with a lethal dose of virulent blood, and third, that the protective properties of the serum only protected those animals which did not react for a period of fifteen days.

I also endeavoured to ascertain whether the serum had any value as a curative agent after the development of the disease, but I regret to state that it has not, as several infected horses, treated at the onset of the disease, when the temperature began to rise, with repeated large doses of serum given both intravenously and subcutaneously, in some cases to the amount of 1,000 cc., failed to rally and died of horse-sickness, although a fall in temperature and the subsequent prolonged course of the disease indicated that the serum did possess some slight therapeutic power.

From this it would appear that horse-sickness serum in its action resembles tetanus and plague serum, both of which have high protective and only slight curative powers.

Fortunately I found that this serum possesses no hæmolytic properties, and does not cause hæmoglobinuria, therefore we are in a position to attempt to immunise animals on similar lines to those which are followed in actively immunising cattle against rinderpest.   What has to be done is to find out what combination of serum and horse-sickness virus will produce a mild attack of the disease sufficient to protect the animal, but not severe enough to

be dangerous. For this purpose we have to ascertain what doses of virus are most suitable; whether it is best to use large doses of virus or merely lethal doses; what dose of serum can be depended upon to control the action of such a virus; whether it is most satisfactory to inject serum and virus simultaneously or with an interval of several days between the injections of each; whether the virus should be given before or after the serum; what is the most suitable site for inoculating, and whether subcutaneous or intravenous inoculations are best. From this it will be seen that there are many important questions still to settle, and answers to these can only be given after experiments have been made.

With a large number of experimental animals these problems could be solved in a comparatively short time, but the number at our command was small, we could only use single animals for each experiment, and several times work was delayed and experiments had to be repeated, as a suspicion always existed that some of the old animals with which we were working might be naturally immune. Under such conditions work progressed slowly, but nevertheless very interesting and practical results are forthcoming.

I have been able to demonstrate that it is possible by combinations of virus and serum to produce in susceptible animals a mild attack of horse-sickness from which they recover, and after which they are immune, and I find that an attack of horse-sickness characterised by no external evidence of indisposition, but merely by a rise of temperature lasting several days, is sufficient to give protection. Up to the present we have artificially immunised three horses and two mules by this method, and these animals subsequently resisted inoculation with lethal doses of virulent blood. Of course they are still to be exposed to natural infection, but our experience in rinderpest and other diseases shows that animals immunised in this manner successfully resist such exposure. Further, it has been found that a dose of serum injected four days after the animal has been inoculated with virulent blood will arrest the development of the disease, and from this line of treatment in particular I expect good results, because the virus has time to develop to a certain degree before it is checked by the action of the serum, and an active immunity results. Three of our artificially immunised animals have been treated in this manner, and now ten horses and one mule have been inoculated in the same way. For economical reasons I began by using relatively large doses of serum to check the disease, and I now propose to diminish the dose of serum gradually until I ascertain what is the minimum dose necessary to control the action of the virus.

Experiments of this description occupy a good deal of time, because a fortnight must elapse in order to allow the serum to be eliminated from the animal's system before testing their resistance to the disease.

Besides these experiments, others are being conducted on lines which answered excellently in rinderpest, i.e,, the simultaneous injection of virulent blood with serum.

The encouraging results obtained, which I have recorded in this report, impress me with the conviction that a practical method of protective inoculation against horse-sickness is within our reach, and I now intend to go on with the experimental immunisation of the shipment of young Argentine horses which arrived here on the 3rd instant, six of which are already under treatment.

This work I hope to complete by the end of March.

SECOND REPORI.[1]

In prosecuting my researches as to the feasibility of artificially producing immunity against horse-sickness, I have found that it is only by the most

[1] This Report is dated 29th March 1904.

scrupulous attention to every detail of the prescribed method that uniformity of results can be ensured. This applies particularly to the preparation of the serum and the virus, which form the basis of the method of treatment by which immunity is established. For this reason, I have added to this Report an Appendix containing a careful description of the manner in which both virus and serum are obtained.

As the serum and virus can be kept for a long time, after the addition of suitable preservatives, without undergoing any change, it is best, before commencing operations, to obtain a sufficient stock of both, in order to make certain that the work is always begun and carried on with virus and serum of a standard strength. Both preparations must be carefully compared with each other, in order that we may be able to regulate and control the action of the one by the administration of the other, and, for economical reasons, the relation between the administered dose of virus and serum should be such, that the virus given should just produce a real attack of horse-sickness, which it is at the same time kept within safe limit by the injection of the minimum amount of serum.

To find these doses, I began by injecting with various doses of fresh and of preserved virulent blood certain horses which we intended to bleed to death, in order to obtain a supply of virulent horse-sickness blood.

The following list gives the result of this experiment :—

LIST I.

A.

				Dose of Fresh Virulent Blood.	Period of Incubation.
No. 1	.	.	.	20 cc.	3 days.
„ 2	.	.	.	15 „	5 „
„ 3	.	.	.	5 „	6 „
„ 4	.	.	.	1 „	10 „

B.

				Dose of Preserved Virulent Blood.	Period of Incubation.
No. 5	.	.	.	10 cc.	5 days.
„ 6	.	.	.	5 „	6 „
„ 7	.	.	.	5 „	7 „
„ 8	.	.	.	5 „	6 „

All the animals in this list developed typical horse-sickness, and showed, on *post-mortem*, the usual characteristic lesions. It was interesting to observe that the incubative stage appears to depend, to a certain degree, on the quantity of virus injected; the animals receiving the largest dose developing the disease soonest, while that injected with 1 cc. of fresh blood had a longer incubative period than any of the others. In this last-mentioned instance, the blood used for inoculation had undergone decomposition, having been kept for thirty-nine days in an ice-chest, insufficiently protected from the air; so that there is some probability that most of the specific organisms which it contained had perished, and only very few had retained their vitality. From this I should infer that an incubation period lasting ten days is practically the longest obtainable by inoculation, for of twenty-eight cases of artificial infection in which the disease was produced in this manner, in only one did I observe a longer period (eleven days), and this occurred in an animal suffering simultaneously from abscess and from nagana (tsetse). Generally speaking, the artificially produced disease has an incubative period of from six to nine days.

Having found from this experiment that 5 cc. of preserved virus was invariably fatal, an experience which bore out the opinion expressed by other investigators, I began a second series of experiments with virus combined with serum. These I carried out on two different lines. In one, serum was given first, and followed by virus. In the other, this process was reverted. The result of the inoculation experiments in which serum was administered before virus was published in my first Report, and here I give particulars of those carried out in the opposite direction :—

List II.

Number.	Dose of Virus.	Interval.	Dose of Serum.	Result.
1 . . .	5 cc.	1 day	120 cc.	Died.
2 . . .	5 ,,	2 days	300 ,,	,,
3 . . .	5 ,,	3 ,,	300 ,,	Severe attack.
4 (Mule) .	5 ,,	3 ,,	200 ,,	Mild attack.
5 (Mule) .	5 ,,	3 ,,	300 ,,	,,

All the animals in this list developed horse-sickness. The two animals which an interval of only one or two days elapsed between the injection of the virus and inoculation with serum, took the disease in so severe a form that death ensued, while the others recovered, and became immune to such a degree that relatively large doses of virus given later produced no reaction whatever. This experiment indicates that the best results are obtained when serum is administered three days after the injection of the virus.

In order to determine whether it would not be possible to obtain equally satisfactory results by the injection of small doses of virus, followed by smaller doses of serum, a procedure which would effect an economy in serum, I then proceeded as shown in List III. :—

List III.

Number.	Dose of Virus.	Interval.	Dose of Serum.	Result.
1 . . .	0·25 cc.	3 days	400 cc.	No reaction.
2 . . .	0·25 ,,	3 ,,	400 ,,	,,
3 . . .	0 2 ,,	3 ,,	200 ,,	,,
4 . . .	0·2 ,,	3 ,,	200 ,,	,,
5 . . .	0·2 ,,	3 ,,	200 ,,	Very mild attack.
6 . . .	0·2 ,,	2 ,,	100 ,,	Fatal attack.
7 . . .	0·2 ,,	1 ,,	100 ,,	No reaction.
8 . . .	0·1 ,,	3 ,,	400 ,,	,,
9 (Mule) .	0·1 ,,	3 ,,	300 ,,	,,

The results obtained in this experiment are more encouraging than those shown in List II. Here, only two animals became sick ; one having a mild, and the other a fatal attack. Both of these were young horses, recently imported, while the other animals dealt with were older, and had all been in South Africa for some time, and none of them showed any visible signs of sickness. Their further immunisation was proceeded with at a later stage of the work. From the outcome of this experiment, I concluded that the individual susceptibility of horses varies greatly, and, as it is necessary to devise a method whereby the most susceptible animal could be safely immunised, I reduced the dose of virus still further, and used only highly susceptible, newly imported Argentine horses for experiment. As I expected that the incubative period would be lengthened by the use of very small doses of virus, I considered it advisable to give the virus more time to act, before restraining it by the administration of serum, and allowed four days instead of three to elapse before giving an injection of serum.

Below I give the results which were now obtained :—

LIST IV.

Number.	Dose of Virus.	Interval.	Dose of Serum.	Result.
1 . . .	0·01 cc.	4 days	200 cc.	Very mild attack.
2 . . .	0·01 ,,	4 ,,	200 ,,	,,
3 . . .	0·01 ,,	4 ,,	200 ,,	,,
4 . . .	0·01 ,,	4 ,,	100 ,,	No reaction.
5 . . .	0·01 ,,	4 ,,	100 ,,	,,
6 . . .	0·01 ,,	4 ,,	100 ,,	,.
7 . . .	0·01 ,,	4 ,,	100 .,	,,
8 . . .	0·01 ,,	4 ,.	50 ,,	,,
9 . . .	0·01 ,,	4 ,,	50 ,,	A somewhat severe attack.

To this list may be added—

10 . . .	0·01 .,	4 ,,	200 ,,	Fatal attack.
11 . . .	0·01 ,,	4 ,,	100 ,,	,,

The death of these last two animals may be attributed to the following causes :—

No. 10 received 0·01 cc. of another sample of virus prepared from the blood of a horse being injected with so-called "passage virus," that is, virus passed artificially through a series of horses probably becoming increased in virulence in the process. The correctness of this conclusion requires to be proved by further experiments with passage virus, but as I could not afford to sacrifice more animals for this purpose, I refrained from using this virus for other experiments.

One deduction which may be drawn from a consideration of List IV., is that not only do animals vary in susceptibility, but various samples of blood vary in virulence, and this is the reason why it is well to obtain a large supply of virulent blood from one animal, and to use it only after its strength has been ascertained by careful experiment.

In the case of Horse 11 (List IV.), the usual method of inoculation was departed from, virus and serum being injected on different sides of the neck, whereas in all other cases the same side was selected for both inoculations. To this cause only can I ascribe the insufficient protection conferred by the serum in this instance, but in spite of this deviation from our customary method, the serum exercised a certain controlling effect over the course of the disease, which was unusually protracted, and for a time the animal looked as if it would recover.

As No. 11 died of horse-sickness in spite of the administration of the usual quantity of serum, while No. 9 had a severe and Nos. 1, 2, and 3 mild attacks of horse-sickness, we may consider that the injection of 0·01 cc. of virus is sufficient to induce a fatal attack of horse-sickness in highly susceptible animals, and as it is very probable that the minimum lethal dose is much less, I would recommend the employment of 0·005 or 0·002 cc. of virus as a first dose for highly susceptible or very valuable animals, while a reduction of the interval between the two inoculations from four days to three will still further diminish the risk. For ordinary purposes, however, I think that the combination of 0·01 cc. of virus and 100 cc. of serum, with a four days' interval between each, may be considered sufficiently practical, as all the animals we inoculated with due attention to the details of the operation withstood this treatment without any serious reaction.

The dose of 100 cc. of serum also appears to be approximately correct, whereas 50 cc. is not enough as it will be noted that of Nos. 8 and 9 which received 50 cc. only (List IV.), one animal became sick, and although it fortunately recovered, a fatal issue might very well have ensued. Later on it may be worth while for economy's sake to try the effect of 75 cc. of serum instead of 100.

The next task was to ascertain whether the animals inoculated as described in List IV. had acquired a degree of active immunity which would enable them to resist further tests. For this purpose all the animals which had been already inoculated with 0·01 cc. of virus received five times that amount —(0·05 cc.), and after a four days' interval only half the former dose of serum—(50 cc.), while a control, a young Argentine horse which had not been previously inoculated, was subjected to the same treatment. None of the animals inoculated previously showed any reaction, but the control animal developed horse-sickness after the usual incubation period, and died.

This goes to show that the preliminary treatment to which these animals were subjected, conferred a certain degree of immunity upon them which was active and not passive, as the second injection of virus was made twelve days after the first serum injection, and consequently after a sufficiently lengthy interval had been allowed to elapse, to ensure the disappearance of any purely passive immunity.

The next stage in the experiments was the injection of these animals twelve days later with 0·2 cc. of virus (four times the previous dose) followed after an interval of four days by an injection of 50 cc. only of serum. No reaction followed, and twelve days afterwards I went on to the injection of virus alone, giving to some animals 0·2 cc. of virus and to others 0·5 cc. These injections of virus produced no reaction, and showed that animals treated in the manner described will acquire a degree of immunity which will enable them to resist inoculation with a dose of virus sufficient to kill from twenty to fifty unprotected animals.

Up to now, fourteen horses have been immunised to such a degree that from 0·2 cc. to 0·5 cc. of virus does not affect them, and in a few days the rest of our animals will be brought up to the same standard, but whether this standard is one which is sufficient to protect them against natural infection has yet to be determined by exposing them in unhealthy localities. If experiment proves that this degree of immunity is insufficient, it is easy to raise their immunity to a still higher pitch, and until they become absolutely immune to all degrees of natural infection. All that will be required for this will be a protracted course of inoculation, while, should it be found that such an artificially conferred immunity proves to be of comparatively short duration under practical conditions, it can easily be maintained by periodical inoculations.

In working with virus uncontrolled by serum, in the later stages of immunisation, my experience has shown that the amount of virulent blood administered must be increased much more gradually than when virus and serum are given with an interval of four days between each. In the latter case, at each successive stage the dose of virus may be increased five or ten times without any risk, but when the stage in the process is reached at which virus alone is given, it is unwise to do more than double the previous dose on each occasion. It may be that this degree of precaution is extreme, and further investigation may show that it is not altogether necessary, but on account of our limited supply of animals, I did not care to push matters too rapidly. Later on, if it is found after exposure to natural infection that larger doses than 10 cc. of virus are required to establish immunity, ordinary preserved virus will not give satisfactory results, as it contains too much glycerine, and recourse must then be had to fresh virulent blood or to blood taken artificially and kept from coagulation by the addition of citrate of potash. Such citrated blood, protected from the air by a covering of paraffin wax, may be kept for months without undergoing any material alteration.

The various stages in the method which I recommend for the establishment of artificial immunity against horse-sickness, which are based upon my recent experience, may be summed up briefly as follows :—

Step 1. 0·01 cc. virus subcutaneously in the neck.   Four days interval.
100 cc. Serum (on the same side subcutaneously, a hand's breadth below the
site of the virus injection).   Twelve days interval.
Step 2. 0·05 cc. virus.   Four days interval.   50 cc. serum.   Twelve days
interval.
Step 3. 0·2 cc. virus.   Four days interval.   50 cc. serum.   Twelve days
interval.
Step 4. 0·5 cc. virus.   Twelve days interval.
Step 5. 1 cc. virus.   Twelve days interval.
Step 6. 2 cc. virus.   Twelve days interval.
Step 7. 5 cc. virus, etc.
It must be borne in mind that the doses given here do not indicate the
quantities of virus and of serum to be given in every case.   They are only the
doses appropriate for use with the samples of virus and of serum with which
we conducted our experiments, and, as virus and serum in all probability
vary in strength in every instance, preliminary experiments will always have to
made with every sample used, in order that a suitable reaction may be
obtained at the first step of inoculation, which is the most important, because
it is at this stage that the foundation of immunity is established, and we found
that if our animals reacted at all they only reacted after the first inoculation.
If the lines laid down in this inoculation programme are carefully followed,
only modifying the doses as may be found necessary, the work of immunising
may be carried out with safety and without loss, but if extra precautions are
considered advisable in dealing with any particularly valuable animal, a pre-
liminary injection of 0·005 cc. of virus followed four days later by 100 cc.
serum, or of 0·002 cc. of virus followed after four days by 50 cc. serum,
should be carried out twelve days before undertaking the first step of the
directions for inoculation.   By the ordinary method which I have recom-
mended, forty-eight days' treatment is necessary to enable an animal to with-
stand 0·5 cc. of virus without injury, while to raise its immunity to a point at
which 5 cc. of virus may safely be given takes thirty-six days more, in all
about three months, but this length of time will only be necessary if no risk is
to be incurred.   If it is desired to shorten the process, however, one stage in
the method may be dropped out, but then of course the process will be
attended by a certain amount of danger.   In several cases, especially in those
in which I observed a slow rise of temperature for some days after the first
step of the immunising process, I ventured to curtail operations by omitting
the third step, and, thinking that the rise of temperature was a sign of a mild
specific reaction, I injected virus without serum twelve days after the com-
pletion of the second stage.   These experiments all turned out satisfactory,
and in this way the time required for immunising was shortened to one month,
but further experiment is necessary before we can state with certainty that Step
3 is superfluous.
I am convinced that experience will show that my method may be simplified
very considerably, but my present task has chiefly been to discover the principal
by which immunity against horse-sickness could be conferred without danger
to the animals treated
I observed, in the course of my observations, that all animals which reacted
severely to inoculation, and developed a characteristic attack of horse-sickness
at once, became highly immune, and resisted injection with 5 cc. of virus.   The
animals in which this occurred (six in number) were immunised in from twenty
to fifty days, but they run more risk under these conditions than is desirable.
It seems to me, that if a rapid method of immunisation is required, an attempt
should be made to regulate the doses of virus and serum in such a way that a
slow rise of temperature occurs in the first stage of inoculation.   Experience
will show, I believe, that this can be done with comparative safety, and then
the whole process will only take from a month to six weeks for its completion,
but, in order to carry on the work with absolute safety, it will be best to com-

mence operations at a time of the year when the disease is dormant, and then animals can be inoculated without danger, and can be brought to a high pitch of immunity before the horse sickness season begins.

In my first report I stated that it was my intention to carry out other experiments, in the hope that by a simultaneous inoculation with virus and serum, working on lines similar to those followed in inoculating against rinderpest, we might also be able to immunise against horse-sickness. For this purpose, I could only spare four horses, and could not, therefore, arrive at any definite conclusion. Inoculation of these four animals was carried out in a similar manner to my other inoculations, serum and virus being injected, for theoretical reasons, on the same side of the neck, the serum injection being a hand's breadth below the site of the virus injection, instead of being made on opposite sides of the body, as in rinderpest.

First, an animal about twelve years old was injected at intervals of twelve days in the following manner :—

Step 1	.	.	.	. 0·2 cc. Virus.	100 cc. Serum.
„ 2	.	.	.	. 0·2 „ „	50 „ „
„ 3	.	.	.	. 0·5 „ „	50 „ „
., 4	.	.	.	. 0·5 „ „	None.
„ 5	.	.	.	. 1·0 „ „	„

This animal did not react. Then three young Argentine horses were submitted to experiment, inoculation in this case also being carried out with twelve days between each.

FIRST STEP.

Horse 1	.	.	. 0·01 cc. Virus.	100 cc. Serum.
„ 2	.	.	. 0·01 „ „	75 „ „
„ 3	.	.	. 0·01 „ „	50 ., „

SECOND STEP.

Horse 1	.	.	. 0·1 cc. Virus.	100 cc. Serum.
„ 2	.	.	. 0·075 „ „	75 „ „
„ 3	.	.	. 0·05 „ „	50 „ „

THIRD STEP.

Horse 1	.	.	. 0·2 cc. Virus.	50 cc. Serum.
„ 2	.	.	. 0·2 ., „	50 „ „
„ 3	.	.	. 0·2 „ „	50 „ „

FOURTH STEP.

Horse 1	.	.	. 0·5 cc. Virus.	0 Serum.
„ 2	.	.	. 0·2 „ „	0 „
„ 3	.	.	. 0·1 „ „	0 „

Treatment will now be continued by the inoculation of 1 cc., 2 cc., and 5 cc., of virus at the usual interval, but the time which has elapsed since the fourth inoculation is as yet too short to enable me to state positively that the animals are not likely to suffer from it, nor am I sure that they will not, because, so far, they have not reacted at any stage of the operation.

If this experiment is brought to a satisfactory conclusion, it will be well to make others, and to attempt to induce a distinct reaction of the first injection by diminishing the amount of serum, or by increasing the dose of virus. Should this recation be obtainable, this method will be preferable to any other, as it is more expeditious, but, if not, more satisfactory and more lasting immunity will probably follow alternate inoculations with virus and serum,

because, by alternate inoculation, more time is allowed for the virus to take effect in the animal organism. Exposure to natural infection will soon prove which method is best for practical purposes.

After determining that it was possible to immunise animals against high doses of virus by means of preliminary inoculation with various combinations of virus and serum, the question arose as to whether such a result might not be attained by the injection of gradually increased doses of virus alone, without the use of serum. Lack of animals prevented me from making many experiments to ascertain this, but I am satisfied that it is really possible to immunise against horse sickness in this manner, as I succeeded in bringing a horse by such method to a stage of immunity which o·1 cc. of virus had no effect upon him, while another withstood o·2 cc., and a third o·5 cc., but it seems that it is necessary to proceed much more cautiously when virus is given than when it is given along with serum. A longer interval must be allowed between the injections, and the dose must be increased more gradually, and, for these reasons, the method has little value from a practical standpoint, although it is of considerable scientific interest.

I will conclude by stating :—

First.—By a combination of injections of virus and serum, it is possible to immunise against horse-sickness, without risk.

Second.—The method which I have recommended can probably be simplified to such an extent that the process of immunisation may be completed in from a month to six weeks.

APPENDIX A.

Directions for the Preparation of Horse-sickness Virus.

The blood is drawn with a canula from the jugular vein of an animal suffering from horse-sickness, after the symptoms of the disease have become quite pronounced. It is then defibrinated, strained through wet muslin, and preserved somewhat after the manner recommended by Dr Edington, 1000 parts of blood being slowly stirred into a mixture containing 1000 parts glycerine and 1000 parts of water, in which 1 part of carbolic acid had been dissolved. Care should be taken to disinfect everything used in the course of these operations, and the water in which the carbolic acid is dissolved should first be carefully sterilised. The preserved virus should then be stored in a deep glass stoppered bottle for two or three weeks in a dark cool place, preferably in an ice chest. During this interval, the red blood corpuscles become dissolved, and the solid constituents of the blood are either precipitated or remain suspended in the solution, and as the virus of the disease is associated intimately with the red blood cells, there is no doubt that much of it is entangled in the precipitate.

Unless this precipitate is separated by infiltration, it is obviously impossible to estimate the exact amount of the dose, and I am convinced that several apparent irregularities in results obtained in the course of our earlier experiments were caused by this fact being overlooked. For this reason, when precipitation is complete, the fluid should again be filtered through wet sterilised muslin. The filtrate should then have the appearance of a dark reddish brown fluid, perfectly clear when viewed against the light, and should not be used unless it is free from any apparent trace of turbidity. After filtering, the virus should be stored in bottles of 50 to 100 cc. capacity. These bottles should be well filled with fluid, should have glass stoppers, and should be kept in an ice box.

To prepare virus for inoculation purposes, sterilised sodium chloride solution should be used (o·85 per cent.) to dilute it, and it should only be diluted as required. In diluting the virus, the solution should be prepared so that 5 cc. will contain the desired dose.

For instance, to obtain a solution containing o·o1 cc. of virus in every 5 cc.

take up 0·02 cc. of virus in a pipette graduated to hundredths of a cc. and mix this with 10 cc. of salt solution ; 5 cc. of this will contain 0·01 cc. of virus.

After drawing the solution into the syringe, it should again be examined to see that it is perfectly clear. The injection is then made in the usual way under the skin of the neck, high enough up to enable the next injection (of serum) being given below it.

<center>APPENDIX B.</center>

<center>*Preparation of Serum.*</center>

After I found that salted animals could resist injections of 20 or even 200 cc. of virulent horse-sickness blood without any ill effect, I went on at once to what may be described as maximum doses, giving subcutaneously as much as two litres of blood drawn from an animal in the last stages of horse-sickness ; but, finding that subcutaneous injections did not raise the immunising power of the serum to a sufficient degree, I adopted the intravenous method of fortification. For intravenous injections, blood must be carefully defibrinated, filtered through muslin, and injected while warm, or, if allowed to cool, it must be raised again to a temperature of 35° C. in a water bath. The blood should then be introduced directly into the jugular with a large canula by gravity alone, and should the respiration of the animal become disturbed during the operation it must be at once discontinued. The first intravenous injections administered I found were borne by the animal without any marked manifestation of discomfort, and I generally gave doses of two litres—in one case going as high as two and a half litres—but I found that after several injections the animals became more sensitive, and the dose consequently had to be decreased.

These injections should not be given at shorter intervals than fourteen days, for in one instance in which we departed from this plan the inoculated animal developed an attack of hæmoglobinuria, the only instance in which I observed this phenomenon in the course of our horse-sickness investigations.

Twelve or fourteen days after the fourth injection the animal can be bled for the purpose of obtaining serum, to the extent of four or five litres. A week later it can be bled a second time, a week later a third, and a week later a fourth. It should then be allowed some months' rest, and should be again fortified.

In our fortification work we only made use of old animals, and this seems to be a point of some importance if serum without hæmolytic properties is to be obtained. If this arrangement is departed from, and young animals used, it is advisable to test every sample carefully before making use of it for inoculation purposes, as there is some probability that serum prepared from the blood of young horses will be found to possess a certain hæmolytic action.

Apart from this question, it is advisable to use old horses for serum work for another reason, as most old animals are immune against biliary fever (equine pyroplasmosis), a malarial disease very common in South Africa. While fortifying our immune animals, I injected intentionally, on several occasions, blood containing the organisms of biliary fever, and never succeeded in inducing an attack of this disease, but one of our young animals inoculated with 20 cc. of blood taken from an old salted horse developed a severe and fatal attack of biliary fever after an incubative period of nine days.

That this disease, biliary fever, may be readily mistaken for horse-sickness, a reference to the existing literature on horse-sickness clearly shows, therefore everyone working on horse-sickness should be quite familiar with the symptoms of biliary fever and with the microscopic appearances presented by the blood of animals suffering therefrom, as this affords the only sure mode of diagnosis. A microscopic search for the specific organism of biliary fever should be conducted with great care, and considerable experience is necessary, as the parasites are not abundant and frequently deviate from the classical pyriform

shape, having an irregular contour sometimes resembling that of the parasites of human malaria. Amongst our animals we had six cases of biliary fever.

As examples of the method in which our salted animals were fortified, I give the following detailed record of the operation as carried out in different cases :—

No. 1.

4/7/03   200 cc. from No.   2 Subcutaneous.
7/8/03 2000 cc. from No.   7 Subcutaneous.
29/8/03 2000 cc. from No. 12 Subcutaneous (contained pyroplasma).
13/10/03 2000 cc. from No. 24 Intravenous.
20/10/03 2000 cc. from No. 16 Intravenous.
                 31/10/03 Bled.

No. 3.

4/7/03 2000 cc. from No.   2 Subcutaneous.
7/8/03 2000 cc. from No.   7 Subcutaneous.
29/8/03 2000 cc. from No. 12 Subcutaneous (contained pyroplasma).
              11/9/03 Bled (Serum too weak).
6/10/03 2000 cc. from No. 14 Subcutaneous.
20/10/03 2500 cc. from No. 16 Intravenous.
              1/11/03 Bled.
3/12/03 2000 cc. from No. 28 Intravenous (contained pyroplasma).
              17/12/03 Bled.
              24/12/03 ,,
              1/1/04   ,,

No. 8.

7/8/03 1000 cc. from No.   7 Intravenous.
29/8/03 1000 cc. from No. 12 Intravenous (contained pyroplasma).
13/10/03 1700 cc. from No. 24 Intravenous.
20/10/03 2000 cc. from No. 16 Intravenous (Hæmoglobinuria).
              2/11/03 Bled.
3/12/03 1700 cc. from No. 28 Intravenous (contained pyroplasma).
              16/12/03 Bled.
              23/12/03 ,,
              31/12/03 ,,
              7/1/04   ,,
24/3/04 1500 cc. from No. 65 Intravenous.

This last dose proved to be too high and the animal died.

After the blood is drawn from the fortified animal, serum is obtained in the following manner :—

The blood, after being defibrinated and strained through muslin, is passed into a deep bottle and set away in a cool place. In twenty-four hours the red blood corpuscles are precipitated, and the supernatant serum can be decanted off. The blood is then set aside, and twenty-four, and even forty-eight hours afterwards, a further supply of serum will be found to have separated. Altogether 70 per cent. of the serum can be obtained from defibrinated blood treated in this manner. The serum is then preserved in the manner recommended by Ehrlich : 10 parts of a mixture made by adding 5·5 parts of phenol and 20 parts of glycerine to 74·5 parts of water being added to every 90 parts of serum. The serum is then passed into 200 cc. bottles, a certain amount of precipitation occurs, and the serum should then be clear and of a reddish colour. Kept in an ice chest it will not undergo any change for a long time.

SOME OBSERVATIONS AND EXPERIMENTS IN CONNECTION WITH TROPICAL BOVINE PIROPLASMOSIS (EAST COAST FEVER OR RHODESIAN REDWATER).

By Dr A. THEILER, Government Veterinary Bacteriologist, and
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I.—EXPERIMENTS TO SHOW HOW LONG AN AREA, WHICH WAS AT ONE TIME INFECTED, WILL REMAIN INFECTED.

IF we consider the fact that East Coast fever is a disease transmitted by ticks, it is quite logical to expect that a pasture upon which infected ticks have been dropped by sick cattle must after a certain lapse of time become clean, provided cattle are prevented from passing over it. We say cattle purposely, because ticks may also develop on other intermediate hosts and so continue their species. We do not, however, attach any importance to hosts other than cattle in the upkeep of infection, since we know that only the bovine species is liable to contract the disease, and the other animals would therefore be unable to reinfect the ticks. We have to conclude, moreover, that an infected area which has been stocked only with other animals than cattle will purify itself all the sooner, because these other species act as hosts for the ticks, which lose their pathogenic properties after sucking on a non-susceptible species. Further, when one takes into consideration (a) the condition under which the disease is spread, viz., by brown and black pitted ticks, (b, that it is only the intermediate stages in

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the life cycle of this tick which take up the infection, (c) that the life time of larvæ, and nymphæ, and adults, must be limited ; then one could reasonably expect that the infection in the ticks on a pasture must die out after a relatively short time if all bovine animals are removed. To ascertain the period of time which must elapse before cattle could be again introduced with impunity on a formerly infected farm, a piece of ground was selected by us near Nelspruit. We had actual proof that at one time the ground was badly infected. On this particular farm the disease had appeared for the first time in May 1902, and by the end of June of the same year every head of cattle had died. It was introduced again in September 1902, and on or about the 15th of that month five oxen died within a certain area over which they had been grazing during the few days previous to their death. Since then no cattle had been on the same farm.

This particular ground was selected for a test. It was marked off from the other portions of the farm by a double fence in order to prevent any possible reinfection of the adjoining ground should the exposed cattle contract the disease. Special care was taken to include within the fence all spots where the sick cattle had died. The fenced area abutted on the railway line, and we were therefore able to introduce cattle from trucks without their having to pass over any portion of suspected ground. On the 5th December 1903, *i.e.*, about fourteen-and-a-half months after the death of the last ox on the farm, ten Texas heifers were brought from Machadodorp and placed in the fenced camp. They were allowed to run without receiving any attention. These cattle came oversea from a country in which East Coast fever is unknown. They, therefore, possessed no immunity, and some of their fellows which were exposed on ground known to be infected died in the usual time. They were received by ourselves from the ship, and were kept inside clean kraals and fed on imported forage previous to their going to Nelspruit. On the 21st January 1904 one of the heifers was found dead in a water hole, and, from the circumstances connected with its death, we had to conclude that it was drowned, probably after having been overpowered by a crocodile. Smears made from the blood of the dead animal did not show any trace of the parasites of East Coast fever. The remaining nine animals were kept inside the fence during the months of December 1903, January, February, March, April, and up to the 30th May 1904, when they were removed by a route over which no cattle had been for more than eighteen months to another fenced-in area. On the latter ground twenty-eight head of cattle had died from Rhodesian tick fever in January and February 1903.

During the same time, Texas cattle from the same shipment were exposed on another farm at Nelspruit, in the neighbourhood of the experimental ground previously referred to. Since May 1903 this second farm had remained infected by constant re-introduction of fresh cattle. Here the cattle died, some of them during the months of December and January.

A final lot of ten Texas cattle were exposed on 7th January 1904, on the same infected farm, and they died during the months following. The results of the latter experiments serve as a control, and prove that the infection did not die out on those places where since the first outbreak (May 1902) cattle had been repeatedly re-introduced.

From the foregoing observations we may conclude that an area which at one time was badly infected with East Coast fever does purify itself again after a reasonably short period. This may be considered for the present to average about fifteen months, but it is possible that our further experiments now in progress may show that a shorter period will suffice for purification. At the same time, we must remark that our general observations on this disease have supplied us with strong evidence that pathogenic ticks may remain active on an infected farm for six months after cattle have been removed therefrom, and we have also a certain amount of evidence to show that infection may last for eight months. The importance of this knowledge is evident, and we are inclined to believe that the necessary period for purification will ultimately turn out to average between twelve and fifteen months.

Similar general observations by Mr Gray regarding the time in which ground purifies itself have since been made in Rhodesia, and they all formed the basis of our recommendations regarding the stamping out policy, which was endorsed by the South African Conference of Veterinary Surgeons in Cape Town, where the following resolution was unanimously carried.

"That this Conference is of opinion that the only effective method of eradicating African Coast fever is to kill off all cattle in infected areas, and to leave such areas free of cattle for a period of not less than eighteen months."

II.—INOCULATION EXPERIMENTS ACCORDING TO THE METHODS OF PROFESSOR KOCH.

In a former article on East Coast fever it was stated by one of us [1] that the injection into susceptible cattle of defibrinated blood of oxen which had recovered from the disease did not produce any reaction, and that the same cattle, when exposed to natural infection, contracted the disease and died. The oxen were, however, only subjected to one inoculation with immune blood. Since then Professor Koch has published some experiments, in which he repeated the inoculation of immune blood every eighth day, and in this way produced what appeared to him to be modified attacks of African Coast fever. He said: "While these experiments have not been numerous, they tend to indicate that the blood of recovered animals is even more suitable for inoculation purposes than that of those which are actually sick." After stating that single small or single large injections of blood from a sick or recovered animal did not afford any protection, he went on to say that repeated injections were more satisfactory, and appeared to confer an undoubted immunity. He further stated his belief that the repeated injection of small doses of blood would confer an immunity which would be heightened in direct proportion to the number of injections to which the animal was subjected, and that his experiments showed that it was not necessary to employ the blood of sick animals for this purpose, as blood taken from recovered animals had a similarly satisfactory effect, and in

[1] Dr Theiler, "Transvaal Agric. Journal."

some cases even a better one. He then recommended that defibrinated blood from recovered animals be injected simultaneously four times, with an interval of seven days between each injection. After this, 10 cc. doses of defibrinated blood were to be given for some time every two weeks, after which one dose a month would suffice.

We decided to follow the recommendations of Professor Koch, and carry out the inoculation as an experiment, under such conditions as would be likely to be met with in an infected area when the disease has begun to make its appearance. We submitted ten animals to four inoculations with 10 cc. of defibrinated blood at intervals of seven days between each injection. We then exposed them to natural infection on the veldt, and continued the inoculations during the time of exposure.

The cattle employed in these experiments came from oversea to the laboratory, and had never been on Transvaal pastures.

Nine Texas calves, Nos. 21 to 25 and 27 to 30, were inoculated as follows :—

1. Inoculation on 28th October 1903.
2. Inoculation on 4th November 1903.
3. Inoculation on 11th November 1903.
4. Inoculation on 17th November 1903.

On 18th November all nine animals were sent by rail to Nelspruit to be exposed on infected pastures. They arrived at Nelspruit on 20th November. The results were as follows :—

Calf No. 21.—The fifth inoculation was made on 1st December. On 2nd December, or after twelve days' exposure, this animal's temperature began to rise. The fever continued for twelve days, and death ensued on 13th December. Blood smears showed a pure infection of East Coast fever. Typical lesions of East Coast fever were present in the organs.

Calf No. 22.—The fifth inoculation was made on 1st December and a sixth on 12th December. This animal started to sicken on 19th January—*i.e.*, after sixty days' exposure. Death took place on 29th January, after an illness of ten days. The piroplasma bigeminum (ordinary redwater) and that of East Coast fever were both found in the blood smears. The infection, then, was mixed in this case, but the chief point is that the animal was attacked with East Coast fever, and typical lesions of the disease were present after death.

Calf No. 23.—The fifth injection was made on 1st December. Fever was noted on 7th December—*i.e.*, after an exposure of seventeen days. Death resulted sixteen days later, on 17th December. Blood smears showed a strong double infection with the piroplasma of East Coast fever and that of ordinary redwater. The lesions were typical of East Coast fever.

Calf No. 24.—The fifth and last inoculation was made on 12th December. The animal was still alive after six months' exposure. Several examinations of blood smears were made during febrile reactions, but the piroplasma could never be found.

Calf No. 25.—The fifth inoculation was made on 1st December and a sixth on the 12th December. Fever set in on 21st December—*i.e.*, after thirty-one days' exposure. The animal died after twelve

days' illness, and both piroplasmata were present in the blood. Typical lesions of East Coast fever were present.

Calf No. 27.—The fifth injection was made on 1st December and a sixth on the 12th December. Fever started on the 18th December —*i.e.* after twenty-eight days' exposure. The animal died after an illness of eleven days. Both piroplasmata were found in the blood smears. Typical lesions of East Coast fever were present.

Calf No. 28.—The fifth injection was made on the 1st December and a sixth on the 12th December. Fever began on 15th December —*i.e.*, after twenty-five days' exposure. The animal died after an illness of fourteen days. The piroplasma of East Coast fever only was present in the blood smears; the lesions were typical.

Calf No. 29.—The fifth and sixth inoculations were respectively made on the 1st and 12th December 1903. Fever started on the 10th January 1904—*i.e.*, after fifteen days' exposure. The disease lasted thirteen days, and ended in death. Both piroplasmata were found in the blood smears; lesions were typical of East Coast fever.

Calf No. 30.—The fifth inoculation was made on the 8th December —*i.e.*, after an exposure of eighteen days. Death took place on 20th December, or after an illness of twelve days. Both piroplasmata were present in the blood smears; lesions were typical of East Coast fever.

Control Experiments.

Along with the nine inoculated calves, five non-inoculated Texas calves of the same shipment were exposed on the 20th November. The results were as follows :—

Texas Calf 31.—Fever began on the 10th January—*i.e.*, after fifty-one days' exposure. The animal died after an illness of eleven days. Both piroplasmata were present in the blood smears.

Texas Calf 32.—Fever began on the 28th December—*i.e.*, after twenty-eight days' exposure. The disease ended fatally after fifteen days. There was a strong double infection with both piroplasmata in blood smears.

Texas Calf 33.—Fever began on 6th December, sixteen days after exposure. Death occurred on 21st December, after fifteen days' illness.

Texas Calf 34.—This animal was killed on 25th December, on account of poverty, after thirty-five days' exposure. It had never shown any reaction, and on *post-mortem* proved to be free from any disease.

Texas Calf 35.—This animal showed fever on 30th December, after forty days' exposure, and died thirteen days later from typical East Coast fever.

Texas Calf 36.—Fever set in on the 3rd December, after thirteen days' exposure. The animal died on the 16th December, after thirteen days' illness.

In all the above cases, with the exception of 34, the typical lesions of East Coast fever were present.

Conclusions.—It will be seen that of the inoculated animals eight died of East Coast fever, while one survived. The average time of exposure before they contracted the disease was thirty days. The non-inoculated animals (the one which was killed after thirty-five days' exposure and which was found to be healthy may be taken

into account) also sickened after an average exposure of thirty days. We must conclude, therefore, that the inoculation, which was repeated six times in the majority of cases, had not produced any immunity. The long time which elapsed before the herd was wiped out must be explained in another way than by accounting for it by the protective effects of inoculation. We have repeatedly observed the same thing in herds upon infected pastures where no inoculation had been tried. There is more than one possible explanation for this apparent irregularity, but at present we are without very exact knowledge. It may be due to a paucity of infected ticks, or, what amounts to the same thing, the parasites for some unknown reason may not always develop freely inside the tick host. We know for certain that hymphæ infected as larvæ do not so surely infect cattle as adults infected as nymphæ. We also know that at certain seasons the ticks do not show the same tendency to bite. Ticks travel but a short distance on their own legs ; the infection of pastures therefore occurs in patches, and one can easily imagine that an animal may graze for months without coming in contact with an infected patch. The same thing is observed in anthrax and quarter-evil. This matter, however, is further dealt with in Article IV.

We regard the survival of Calf 24 as a simple coincidence, considering that the mortality of this disease is about 95 per cent., and we have seen exactly the same thing take place repeatedly where inoculation did not occur as a factor to be considered. We admit that in the face of Dr Koch's latest statement, "that immunity can only be expected after five months, during which the inoculation has to be kept up," the foregoing experiments prove nothing against his latest assertion. The experiments show, however, that on infected ground the inoculation would be of little value, since the animals under treatment died off as rapidly as did the controls, and would die off during the immunising process.

The experiment of repeatedly inoculating cattle according to Dr Koch's pronouncement during five months was undertaken with sixteen animals. During this period they were injected thirteen times, and were kept on non-infected ground.

The exposure took place only at the end of June 1904, so that results cannot yet be expected.

The question of immunity, however, after that time has, we think, been settled by the trials in Rhodesia, the results of which were communicated to the South African Conference of Veterinary Surgeons at Cape Town by Mr Gray. The Conference recorded its opinion in the resolution already alluded to, and by another as follows : "That this Conference, after considering the reports of the scientists who have had practical experience of the effects of inoculation as proposed by Dr Koch, is reluctantly compelled to conclude that it will be vain to trust to inoculation to arrest the spread of African Coast fever."

III.—DIPPING EXPERIMENTS.

Although at the time when East Coast fever first appeared in the Transvaal, the particular species of ticks which could communicate the disease were not known, yet everything pointed to the fact that the disease must be carried by ticks. Accordingly, it was thought

that by destroying ticks the spread of the disease might be arrested. From the very first appearance of the disease, advice was given to dip or wash the cattle with anti-parasitic fluids, and then remove them to fresh non-infected areas. In this way many herds were freed from the disease, but this was probably due more to changing the pastures than to the dipping as such. It was, indeed, observed that changing the pastures without any dipping was followed by similar success, whereas dipping without change of pasture did not seem to stop the outbreaks in infected areas. There was much controversy about the effects of dipping in infected areas, and also regarding the effectiveness of the dips used. As no experimental evidence was available regarding the efficacy of dipping as a preventive for East Coast fever, we decided to carry out some experiments with different dips in a badly-infected area, in order once and for all to settle the points in dispute. The Anglo-American Stock Trading Company kindly handed over to us a number of imported Texas cattle, derived from parts of that country known to be badly infected with Texas fever. Their object was to ascertain whether their animals would prove to be immune against East Coast fever. The cattle to be experimented upon were divided into lots of ten ; one lot was to be dipped regularly, and the other was to serve as a control, that is, they were not to be dipped at all. The experiment started on 22nd October 1903, at Nelspruit, on a farm which we knew from previous experience to be badly infected. All the cattle had up to that date been kept in a clean kraal in Machadodorp, and had been fed on imported lucerne only. They were brought to Nelspruit by rail, and directly after off-loading from the trucks they were sprayed by means of a hand-spray. In order to obtain through soaking of the skin, the animals were made to lie down. The spraying was repeated every eight days. The materials used for dipping were : (a) paraffin, (b) arsenical solutions, (c) izal and arsenic.

1. *Paraffin finely divided in water.*—The mixture was obtained by a spray pump called " The Success," and the proportion of paraffin to water was one to four—25 per cent. paraffin. Two animals were treated in this way, Nos. 7 and 10.

Result.—No. 7 came into fever on the 5th November, viz., after fifteen days' exposure ; it had been sprayed twice during that time. The fever lasted up to the 18th November—thirteen days, when the animal died of typical East Coast fever. The infection with piroplasma was a very strong one.

No. 10 started to react after the same period of exposure, and died on the 15th November, *i.e.*, after an illness of ten days' duration. This animal had also been dipped twice during the exposure. On the day of death about 50 per cent. of all red corpuscles were infested with piroplasma.

Conclusions—Taking into consideration that on the date of the first dipping no pathogenic ticks could have been on the cattle, and that the incubation time of the disease averages twelve days, we came to the conclusion that the dipping did not materially retard the infection. A careful examination was made every day after dipping. It was found that only a few ticks were present on the upper parts of the body, and that most of them were dead. On such parts, however, as the tip of the tail, the heels, and in between the hoofs, living ticks

could constantly be found. The brown tick was at that particular period of the year conspicuous by its scarcity.

2. *Arsenical Dips.*—Demuth's modification of the Queensland dip was used. This modification allows the material to be used in solution with cold water, whereas the original formula had to be dissolved in warm water. The solution was made 1 to 25 of water, it being considered by the maker that in a solution of 1 to 30 the efficacy would be the same as that of the original formula. The dip was applied by means of the Douglas spray pump. Six animals were thoroughly sprayed in the way before indicated.

Result. Texas Calf No. 3.—Fever began on the 5th November, after fifteen days' exposure to infection. During this time the spraying was carried out twice. The animal died on the 19th November, or after an illness of fourteen days. The infection with piroplasma of Coast fever was a very strong one.

Texas Calf No. 4.—This animal also became sick after fifteen days' exposure, and it died after an illness of fourteen days. Piroplasmata were very numerous on the day of death.

Texas Calf No. 5.—This animal sickened after an exposure of twenty-five days, and it died of East Coast fever after an illness of fourteen days.

Texas Calf No. 6.—The reaction started on the fifteenth day after exposure, and death resulted fourteen days later, on the 19th November. There was a strong infection with piroplasmata.

Texas Calf No. 8.—This calf started to show fever after forty-seven days' exposure. The disease lasted fourteen days. The animal died on 19th December with a strong infection of the red corpuscles. The calf was sprayed four times, when the operation had to be discontinued on account of the skin being scalded.

Texas Calf No. 9.—The fever began on the fourteenth day; death resulted on the 15th November 1903, after an illness of eleven days.

Conclusions.—Out of six animals which were sprayed with arsenical solution, two contracted the disease after fourteen days, two after fifteen days, one after twenty-five days, and one after forty-seven days. In four animals the dipping had no marked effect; it did not retard the infection. It apparently did so to slight extent in one case, and distinctly in the case of No. 8. But it must be remembered, as we have already pointed out, that some animals which are not dipped escape infection for a much longer period than others, and therefore their survival may be due to other causes than dipping.

3. *Experiments with arsenical dip and an addition of 10 per cent. raw izal.*—The mixture was also applied with a Douglas spray pump. Two animals were treated, Nos. 1 and 2.

Result. Texas Calf No. 1.—Sickened after an exposure of fourteen days, and died after an illness of nine days, with a strong infection of Rhodesian tick fever piroplasmata.

Texas Calf No. 2.—Died from East Coast fever after an exposure of twenty-four days and an illness of fifteen days.

Spraying every eight days with arsenical solution in the strength used so blistered the skins of the animals that it could not have been kept up indefinitely.

Conclusion.—The addition of izal did in no way increase the effectiveness of the arsenical dips.

Result with the (non-sprayed) control animals exposed at the same date.

		Time of exposure.		Disease lasted.
No. 11	. .	25 days	. .	15 days
No. 12	. .	12 days	. .	16 days
No. 13	. .	12 days	. .	16 days
No. 14	. .	14 days	. .	11 days
No. 15	. .	14 days	. .	12 days
No. 16	. .	14 days	. .	12 days
No. 17	Died 21 days later from exhaustion.			
No. 18	. .	10 days	. .	10 days
No. 19	. .	24 days	. .	14 days
No. 20	. .	28 days	. .	17 days

A comparison of the average incubation time in the sprayed cattle with the average incubation time in non-sprayed cattle should give some indication of whether the dipping can be considered to have warded off infection. It averaged in the first instance sixteen days (out of ten cases), and in the second instance seventeen days (out of nine cases). Thus it appears that dipping in a badly-infected area has no effect whatever if the animals continue to be exposed to infection; the non-dipped animals had even a longer average incubation time than those dipped. Although the average incubation time does not demonstrate that the dipping exerted any influence, it might be argued that, if the shortest periods of incubation are taken into consideration, then a slight inhibitory influence is noticeable in some instances. It is, however, short enough to be negligible, as at the utmost it can only be calculated at a difference of one day.

There is also an apparent slight variation in the duration of the disease in dipped and non-dipped cattle. The periods of sickness amounts to an average of 12·8 days in the former, and to 13·3 days in the latter. The difference, however, we consider too small to justify the conclusion that the dip had an influence on duration of the disease.

For practical purposes we consider that we have demonstrated that the application of dips in a badly-infected area gives no guarantee against the further spread of the disease, and no hope of extinguishing an outbreak unless the animals be also removed to fresh pastures. The latter operation, however, incurs the serious risk of infecting the route over which the animals are removed, not to speak of the new pastures.

In coming to these conclusions we do not wish it to be understood that we are foes to the general idea of dipping cattle. We agree with those who hold that systematic dipping will reduce the number of ticks on a farm, but it is principally the blue ticks and those with a similar life history which are affected. The blue tick remains on an animal for the best part of a month, whereas the intermediate stages of the brown tick may drop off in three days.

The dipping materials at our disposal do not in our experience keep ticks off for any appreciable time, and it is not practical to dip cattle say every fourth or fifth day in an irritating and poisonous bath.

We are also far from advising private individuals against erecting

a dip on their farms for the exclusive use of their own cattle. The brown tick, on account of its short sojourn, is difficult to catch by the shortest possible periodical dipping. It is only, however, reasonable to suppose that a few will be caught and destroyed at each dipping, and that years of the dipping *régime* will reduce the number of brown ticks on a farm ; but we doubt if the reduction will be a very material one, and we certainly do not think that anything approaching annihilation will be accomplished, for it must be remembered that many other animals besides cattle act as hosts for the development of the tick in question, although they do not infect it with disease.

We are of opinion that those who are inclined to trust to dipping to keep tick fever off their farms are leaning on a broken reed. If a man fences a clean farm, keeps his cattle on the place, and only brings in fresh animals after they have undergone a period of quarantine for six weeks in a shed or special paddock, we do not think he need greatly fear tick fever even in an infected district. If, however, he insists on doing transport with his oxen on dangerous roads, his animals will sooner or later pick up the disease and bring it on to his farm in spite of dipping.

In conclusion we wish to express the opinion that the plan of erecting a few common dips in a district for the use of all and sundry is calculated to spread the disease, as the pastures around these dips are likely to become infected.

IV.—POSSIBLE INFLUENCE OF THE DIFFERENT SEASONS ON THE OUTBREAKS OF EAST COAST FEVER.

During the years 1903 and 1904 several lots of cattle were exposed at Nelspruit, which has been repeatedly alluded to as a very badly infected area. The number of animals exposed in the different experiments amounted to eighty-nine, of which, so far, only one has recovered. The different lots were exposed during different months of the year. We observed that the shortest average time required to wipe off a lot of twenty-three cattle was twenty-two days and nineteen hours. This is the shortest period on record, and may serve for comparison with the periods after which other lots died during the various months. We obtain in this way some indication of the influence the season may have on the development of the disease.

First Lot.—Exposed on the 14th January 1903, twenty-three animals. Time of exposure before last animal died, twenty-two days and nineteen hours.

Second Lot.—Exposed on the 8th March 1903, seventeen animals. Average time of exposure before death, 24 days 11 hours.

Third Lot.—Exposed on the 3rd April 1903, four animals. Average time of exposure before death, twenty-five days and eighteen hours.

Fourth Lot.—Exposed on the 25th July 1903, six animals. Average time of exposure before death, thirty-nine days sixteen hours.

Fifth Lot.—Exposed on the 22nd October 1903, nineteen animals. Average time of exposure before death thirty-one days seven hours.

Sixth Lot.—Exposed on the 20th November 1903, thirteen animals. Average time of exposure before death forty-four days three hours.

Seventh Lot.—Exposed on the 7th January 1904, eight animals. Average time of exposure before death twenty-five days and ten hours.

Conclusions.—The shortest period required to wipe out an exposed herd of cattle numbering twenty-three head is about twenty-three days. This happened in the months of January and February. During the following months, March and April, the periods vary only by one or two days. The delay begins in June and is noticeable up to the end of the year. The longest period of exposure required was November and December. It is rather a startling observation that the infection was so long retarded in these two months during which the temperature is so favourable for the development and moulting of ticks. A careful examination of the cattle. however, during those periods was constantly made, and it revealed the remarkable fact that the brown ticks were the reverse of plentiful. The veldt had been burnt during the winter. During the month of October the grass had hardly sprung up, and it was very short during the following months. As soon as the grass had fully grown the ticks became more frequent, and were then found to be present in the ears in large numbers. The experience at Nelspruit seems, therefore, to indicate that the rapidity of infection is in direct relation to the growth of the pasture. This might be explained by the observation that the ticks, in order to reach a host, climb to the tops of grasses from which they have every chance of attaching themselves to animals ; hence their scarcity on cattle when the grass is short, and their frequency when the grass is long. We may further conclude that the greater the number of ticks which infest a beast the more is the chance of their being pathogenic ones amongst them. Accordingly, the chances of rapid infection are greatly increased when the grass is long. This probably helps to explain the common observation that grass burning has a decided influence on those diseases which are carried by ticks, but, of course, many of the carriers must be destroyed by fire. We would like to state, however, that this observation made at Nelspruit in the Low Country does not altogether correspond with the number of outbreaks occurring all over the Transvaal, but it must be pointed out that the veldt is not burned at the same time in each district, nor are the other circumstances exactly similar.

INOCULATION AGAINST AFRICAN COAST FEVER.

By C. E. GRAY, M.R.C.V.S., Chief Veterinary Surgeon, Rhodesia.

SHORTLY after the publication of Dr Koch's third report upon African Coast fever, containing his recommendations for inoculation against that disease, the Rhodesian Government, finding that these recommendations were not looked upon favourably by the majority of stockowners, decided to test the method on a somewhat large scale, with a view to determining whether the benefits derived therefrom

were such as to justify legal measures being taken to enforce its adoption, and the Victoria district of Mashonaland was selected as the most suitable field for commencing operations.

This district, which contains a large native population owning approximately about 60,000 head of cattle, had become infected with African Coast fever through the agency of transport cattle working on the Victoria-Selukwe road about a year previously, but owing to the embargo placed upon stock movements the disease had spread so slowly that a circular area, having a diameter of 25 miles around the township of Victoria, contained within its limits nearly all the kraals at which the disease had made its appearance, and a considerable number which were clean but which were in dangerous proximity to infected veldt.

The principles of inoculation as laid down by Dr Koch were explained to the natives by the Native Commissioners, and, as a majority expressed themselves as willing to allow their animals to be treated, the work was begun in the month of November 1903, under the supervision of Dr Koch himself, the recovered animals used to provide the salted blood for immunisation being selected by him as suitable for this purpose. The prosecution of experimental work, however, at Bulawayo, which required his personal attention, prevented Dr Koch from remaining at Victoria during the whole course of the work, but after his departure it was entrusted to competent men specially trained to carry it out, and these men reported progress at regular intervals until the end of May 1904, by which time the cattle at the infected and adjoining kraals had been fully inoculated in accordance with Dr Koch's directions, and then the work was suspended, as it was considered that the benefits of the treatment should by that time become apparent.

For the information of those who have not procured Dr Koch's reports, it may be explained here that this scientist stated that although African Coast fever could not be communicated in an acute form to susceptible animals by inoculation with blood drawn either from animals suffering from the disease, or from those which had recovered from it, still repeated inoculations with blood taken from sick or recovered animals was not without effect, and after several inoculations peculiar ring-like parasites were found in the blood of inoculated animals upon microscopic examination. These parasites he considered to be peculiar to African Coast fever, and he gave it as his opinion that their presence indicated that animals in whose blood these were found had been exposed to infection and were in all probability more or less immune. He also expressed the opinion that repeated inoculations with recovered blood persisted in over a long period would ultimately protect animals so inoculated against acute and fatal attacks of the disease.

In his third report, Dr Koch advised subcutaneous inoculation of susceptible animals with 10 cc. each of fresh defibrinated blood taken from the jugular vein of an animal which had recovered from the disease—inoculation to be carried out once a week for four weeks, then once a fortnight for two months, and afterwards once a month.

This line of treatment he modified in his fourth report to the extent of recommending regular fortnightly inoculations with 5 cc. of defibrinated blood for a period of four or five months.

Inoculation in the Victoria district was conducted strictly in accordance with these recommendations, and when the work was stopped at the end of May over 3700 head of cattle had been subjected to twelve inoculations, and close on 2000 head to eight or nine.

During inoculation and since it was stopped all inoculated herds have been kept under observation, and the subsequent progress of the disease has been carefully noted. I regret to say that while Dr Koch's prediction that herds infected at the time when inoculation was begun would not be benefited thereby has been fulfilled, neither have those herds been protected which were clean at the time we started, nor has the percentage mortality in such herds been diminished when the disease appeared. For example, a herd of 74 head which remained healthy up to the date of the eighth inoculation, and which was inoculated twelve times, has now been reduced in number to 43, and the animals are still sickening. In another case, in a herd of 39, inoculated eleven times, the disease appeared after the tenth inoculation, and now only 13 are left, many of which are sick ; while in yet another herd, formerly 100 strong, and healthy, after twelve inoculations the disease has now broken out, 6 animals being sick and 1 dead.

These are a few of the more noteworthy instances in which inoculation has failed to protect, and their number might be added to were this necessary, but I think that those mentioned indicate sufficiently that repeated inoculations with the blood of recovered animals, as recommended by Dr Koch, has failed to afford the protection against African Coast fever which we all hoped would follow such treatment. It does no harm, it does not communicate the disease, but it fails to protect, therefore I cannot conscientiously recommend the public to depend upon such a method of inoculation, nor yet upon modifications thereof which have been suggested and practised by those quacks who have followed in Dr Koch's footsteps, and who claim to have inoculated successfully against African Coast fever because no mortality has followed their operations, or because there has been an apparent cessation of the disease after inoculation has been carried out. Such apparent checks in the progress of the disease often inspire false confidence in the individual whose cattle have been treated, and lead him to voice the opinion that such and such a method is " all right," but, unfortunately, these checks appear to be due not to any protection conferred by inoculation, but to the temporary absence of infecting ticks, and sooner or later the disease reappears unless the animals are removed from the infected veldt, with the ultimate result that only 4 or 5 salted animals survive out of every 100 which become infected.

The failure of Dr Koch's method leaves us at present with no other weapons to combat the progress of the disease than those which previous experience has dictated, viz., the suspension of movement of cattle so far as is possible, fencing, systematic dipping or spraying (which must, however, be kept up for at least two years before any benefit will be apparent), and the exclusion of all cattle from infected areas for a period of at least fifteen months, in order to give the veldt time to become clean again.

TSETSE–FLY DISEASE AMONG MULES IN THE SUDAN.

By A. S. HEAD, Lieutenant, Army Veterinary Department, Khartoum.

OF the many troublesome and fatal diseases that attack nearly every description of domesticated animal in Africa, making the development of the country a thing nearly impossible owing to the dearth of transport animals and animals for food supply, perhaps none has caused such loss to the early settler and explorer as the tsetse-fly disease.

Tracts of good fertile country have had to be abandoned owing to the impossibility of getting transport animals to and from them through a belt of country infested by the tsetse-fly.

Lately this disease has been brought very prominently before the public owing to the discovery that human sleeping-sickness is also caused by a trypanosoma, and that a fly is the instrument of infection. The disease extends in numerous belts from South Africa to the Sudan ; in fact, it is found in nearly every marshy place where wild animals abound.

With the increase of civilisation and the decrease of wild animals, and the cultivation and drainage of the land, the disease will no doubt be pushed farther and farther away, and in time become a disease of the past.

The disease is caused by a trypanosoma which in the later stages of the disease can be found in large quantities in the blood stream, but in the early stages it is often impossible to find the trypanosoma in the peripheral blood. It affects all domesticated animals, and is nearly always fatal. The only known carrier of the disease is the tsetse-fly.

The tsetse-fly resembles in size the small house fly, but, when resting, its wings close over each other like the blades of a pair of scissors. It has a yellowish thorax marked with longitudinal dark lines, a yellowish white abdomen of five rings, and a long proboscis.

It attacks animals and man by day, but fortunately only rarely by night, so that it is possible to get animals without very much danger through an infected belt provided the travelling is done by night and high ground is reached before daylight.

The fly is said to disappear from a locality with the disappearance of big game, and this has given rise to the theory that wild animals are the host of the trypanosoma without having their health affected by it, and that the tsetse-fly living on their blood infects any domesticated animal it may happen to bite.

How far this theory is correct, or what part the tsetse-fly plays, whether only as a carrier, or as an important agent in the life history of the trypanosoma, are points still to be investigated. The natives have great faith in the dried and powdered tsetse-fly, given internally, as a preventive against the disease.

Symptoms.—The first thing that is noticed is that the animal becomes thin and listless, while its coat loses its gloss. This is followed by progressive anæmia, great debility, œdema of the sheath and

extremities, hanging head, drooping ears, and running from the eyes. The animal has a sleepy look but eats well. Temperature high—102° F. to 104° F. Urine and fæces normal. The period of incubation seems to vary from two weeks to two months, and death may be delayed for weeks and in some cases months. A positive diagnosis can only be made when the trypanosoma can be found in the blood.

Post-mortem.—Mucous membranes anæmic. Gelatinous yellow œdema of the subcutaneous connective tissue, sheath, and extremities; also around the base and auriculo-ventricular furrows of the heart.

Mule affected with tsetse-fly disease.

The whole carcase shows signs of emaciation, but all the organs appear normal.

In one case I found slight inflammation of the stomach, which could not be attributed to drugs.

The tsetse-fly was found, I believe, for the first time last year in the Bahr-el-Ghazal (Sudan) by Lieut.-Col. Griffith, D.S.O., P.V.O., Egyptian army.

The following is the history of six mules which left Khartoum and went to the Bahr-el-Ghazal.

On 1st January 1904 six Syrian mules left Khartoum by steamer for the Bahr-el-Ghazal province, landing at Nashra-el-Shol, and travelling *viâ* Tonj to Nian Niam, afterwards doing transport work between the following places: Tonj, Rombeh, Darago, and Shambe. They returned from Shambe to Khartoum by steamer, arriving 1st June 1904.

They all seemed in good health on arrival at Khartoum, except for being thin and showing signs of travel.

On 1st July 1904 Mule No. 1 began to show symptoms of pro-gressive anæmia; it became listless, with drooping ears and hanging head, discharge from the eyes, high temperature, and œdema of the sheath.   It died on 7th July 1904.   No *post-mortem* was made.

On 10th July 1904 Mule No. 2 was admitted to hospital showing the same symptoms.   Treatment—soft food and tonics.   It died 16th July 1904.

Post-mortem showed gelatinous yellow œdema around sheath and auriculo-ventricular furrows of the heart.   The whole carcase was very emaciated.

On 13th July 1904 Mule No. 3 was admitted to hospital for emacia-tion and debility, which progressed rapidly, the animal becoming very anæmic, standing with head hanging down, ears drooping, and slight discharge from the eyes, œdema of sheath and legs; appetite good. The animal had the appearance of being asleep the whole time.   On the 29th I took a blood smear, and two trypanosomata were found in it.   Later in the day several other blood smears were taken, and the trypanosomata were found in the blood in large quantities.   This animal died in the evening of 29th July.

Post-mortem.—Coat staring, visible mucous membranes anæmic, œdema of sheath and extremities, great emaciation.   All the organs healthy, with the exception of yellow gelatinous exudate around the sheath, and around the base and auriculo-ventricular furrows of the heart.   No trypanosomata were found in two smears taken from the spleen and spinal fluid.

On 21st July 1904 Mule No. 4 was admitted to the hospital suffer-ing from progressive anæmia.   There were no very marked symptoms in this mule, except progressive anæmia.   He fed well, and had a temperature of 101°, until 29th July, on which day a blood smear was taken but without any trypanosomata being discovered.   On 30th July this animal suddenly took a turn for the worse.   Œdema of the sheath and legs appeared, temperature 104° F., pulse small and feeble, and respiration hurried.   The animal had the look of being asleep, with hanging head and hind legs propped together.   Urine clear; feeding well.   Blood smears were taken, and trypanosomata were found in large quantities.   The symptoms progressed, and the animal died on 4th August 1904.   Trypanosomata were found in blood from 30th July until the day of death.

I was, unfortunately, away when this animal died, but understand that nothing abnormal was found at the *post-mortem*, except the gelatinous œdema of the sheath.

Mules 5 and 6 are apparently in very good health.   I have con-stantly examined their blood, but have found nothing.

I am indebted to Dr Christopherson for his help while searching for the trypanosomata in these cases, and also for the photograph of the mule.

A TRYPANOSOMA FOUND IN BLOOD OF CATTLE IN INDIA.

By CAPTAIN DURRANT M.R.C.V.S., A.V.D., and CAPTAIN J. D. E HOLMES, M.A., M.R.C.V.S., C.V.D., INDIA.

A TRYPANOSOMA having the characters shown in the accompanying diagram (Plate IV.) was recently observed at the *post-mortem* of a bull used for experimental purposes at Muktesar Laboratory.

A small bull of Hill Breed, weighing about 250 lbs. and aged five years, was used with others for the purpose of testing the potency of a bulk of anti-rinderpest serum. This bull got a very slight attack of rinderpest accompanied by a loss of condition from which he appeared to be recovering. A week after the rinderpest reaction had passed over, and during which the bull fed well, other symptoms appeared— diarrhœa, intermittent fever, loss of appetite, staring coat, anæmia. The bull died six days later. From the appearance of the secondary symptoms we suspected the complication of piroplasmosis, and on two different occasions examined films of blood stained by a modification of Romanowsky's method. There were many signs of an anæmic condition, the red corpuscles were pale, many with crenated edges, numerous poikilocytes, increased number of esinophiles, and many erythrocytes showing basophile granulations. The latter condition I have observed in almost every instance when examining blood from cattle recently recovered from rinperest. No piroplasmata were found.

At the *post-mortem* nothing further than a general anæmic condition of the body was observed. On examining a fresh smear from the spleen, several living trypanosomata were seen in each field. At first it was suspected that the bull might have been inoculated with horse surra by means of the tabanidae which at this time were prevalent in large numbers. We had several experimental animals (ponies, rabbits, and guinea pigs) suffering from surra, and on several occasions had found the blood of these horse flies teeming with trypanosomata.

However, after staining some specimens taken from the spleen with double stain, it was seen that this trypanosoma varied in many respects from that of horse surra. It is 2 to 4 μ shorter, the body is much thicker, and the posterior end blunter. The centrosome is situated much nearer to the nucleus, and not close to the posterior end as in the trypanosoma evansi. The nucleus and flagellum are more difficult to stain.

This trypanosoma appears to us to more closely resemble the form described by Theiler as affecting cattle in South Africa [1].

Unfortunately, inoculation experiments were impossible owing to the decomposed condition of spleen and blood.

The observance of this trypanosoma has many points of interest. Up to the present it has not been demonstrated that the cattle of India suffer from trypanosomiasis. It is known that cattle experimentally inoculated with the trypanosoma evansi are not affected. After inoculation the trypanosoma appears in the blood in small numbers, at

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[1] "Journal of Comparative Pathology and Therapeutics," September 1903.

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intervals, for two or three weeks, after which it entirely disappears, and the health of the animal is in no way affected.

It has also been found that the blood of a bull inoculated with horse surra, and from whose blood the trypanosoma has for many months been absent, is still capable of conveying the disease to other susceptible animals—horse, rabbit, guinea-pigs, etc. It has yet to be ascertained if the trypanosoma herewith described is capable of causing any grave affection among cattle, or whether, like the trypanosoma evansi, it simply lies dormant in the system. In the case under description there is no doubt that, after the normal resistant power of the cells had been reduced by the attack of rinderpest, the trypanosoma assisted in bringing about a fatal termination.

No clinical symptoms ascribable to trypanosomiasis have been recorded in India, but this is no argument against the existence of the disease.

Until recently it was not known that the cattle of this country were infected with piroplasma.

In 1902 Stockman[1] demonstrated the pear-shaped bodies in the blood of cattle being used for rinderpest experiments. Since then further investigation has shown that a very large percentage of cattle are affected, and, as far as our present knowledge goes, piroplasmosis by itself causes no symptom of illness in Indian cattle, but rather plays a secondary part when the animal is attacked by another debilitating disease.

In the Veterinary Record, 30th August 1902, there is an extract from an article by Laveran and Nocard describing a very fatal outbreak of surra among the cattle of Mauritius. The outbreak was ascribed to surra introduced by cattle imported from India. It is very improbable that this outbreak was caused by the trypanosoma evansi, which has never been noticed in cattle of India except when experimentally inoculated.

DESCRIPTION OF PLATE IV.

Full grown and young forms of trypanosoma obtained from spleen of bull. Magnification 2500. Drawn with oil imm. apochromatic 2 mm. lens. Ocular No. 8 compensating. Enlarged by photography one and a half and redrawn.

EVOLUTION OF THE TRYPANOSOMA EVANSI.

By J. D. E. HOLMES, M.A., M.R.C.V.S., Capt. I.C.V.D.,
Assistant Bacteriologist, Muktesar, India.

ONE of the greatest obstacles to the furtherance of this study is the difficulty of following in a fresh specimen the various stages through which the parasite passes. Most observers agree that it is in coloured preparations only that the evolution of this hæmatozoon can be suitably followed. The many improvements in the methods of staining, principally that of Romanowsky, and the various modifications of his method, introduced by Laveran, Ziemann, Nocht, and others, have greatly facilitated an investigation of this kind. There

[1] "Veterinary Record," 11th April 1903.

still remain, however, many sources of error and many gaps to be filled, so that the interpretation to be given to the phenomena as illustrated by the staining process must vary to a large extent with the individual worker.   This is very evident from the literature already published on the morphology and reproduction of the trypanosomata.

Rabinowitsch and Kempner [1] have recorded the result of their investigation of the life history of the trypanosoma lewisi.   They describe three forms of division : (1) longitudinal, (2) transverse, (3) segmentation.   In the latter case the flagellum and undulating membrane disappear, the parasite takes a spherical form, and the nucleus, dividing several times, gives birth to a number of young elements.

Danielewsky [2] distinguishes two forms only of multiplication : (1) longitudinal division, (2) segmentation.

Wasielwski and Senn [3] argue that all division is longitudinal, as among the other flagellata, and that the mother cell is always recognisable by its larger size.

Laveran and Mesnil [4] describe methods of reproduction in the following stages : (1) the trypanosoma enlarges, the nucleus and centrosome increase in volume, and the centrosome takes an elongated form ; the base of the flagellum thickens, and finally the centrosome and nucleus approach each other.   The nucleus and centrosome divide.   There is no rule as to which divides first.   With division of the centrosome the base of the flagellum divides, and the newly formed branch separates from the old.   The new flagellum lengthens, the protoplasm divides, and a young form separates from the mother cell.   The young parasite may again sub-divide before separation, and two or more young forms appear attached to the parent cell. (2) The method of multiplication of this group differs from the above in that the mother trypanosoma is not distinguishable.   The parasitic elements present varied forms, round, spherical, or irregular.   One sees a number of centrosomes and a number of nuclei.   From the centrosomes are short flagella.   When the number of centrosomes and nuclei are equal the protoplasm divides into a similar number of young forms, each containing a nucleus, centrosome, and flagellum. This description corresponds largely with the longitudinal division and segmentation forms of Rabinowitsch and Kempner.

The trypanosoma brucei has been studied by Plimmer and Bradford.[5]   They have seen the longitudinal and transverse methods of division described by Rabinowitsch and Kempner as taking place in the evolution of the trypanosoma lewisi.   They also describe a process of conjugation.   Two parasites unite with their micro-nuclei in close apposition and with more or less of their bodies fused together.   This is followed by a breaking up of the chromatin into granules and the formation of amœboid forms.   These amœboids possess the power of again segmenting, and finally develop into flagellate organisms.   They frequently unite to produce plasmodial forms.

1 "Zeitschrift für Hygiene," Bd. XXX., 1899, p. 251.
2 "Arch. Slaves de Biologie," 1886-87.
3 "Zeitschrift für Hygiene," 1900, Bd. XXXIII., p. 444.
4 "Annales de l'Institut Pasteur," 1901, p. 675.
5 "The Veterinarian," Vol. LXXII., p. 648.

Laveran and Mesnil[1] are not in accord with these views. They argue that the trypanosoma brucei multiplies by longitudinal division only. They do not admit the conjugation form. It is probably a stage of longitudinal division or due to abnormal conditions, as it is never seen in the fresh blood. They consider the amœboids to be an involution process produced by conditions unfavourable to the life of the trypanosoma. They have not seen plasmodial forms.

Elmessian,[2] who has studied the parasite of Mal de Caderas, describes multiplication by direct longitudinal division only. He has not seen transverse division.

Brauer[3] records his observations on the trypanosoma of surra. He doubts the correctness of the methods of multiplication described for the other trypanosomata, and believes that reproduction is by means of spore formation. The nucleus changes into a coccus about $\frac{3}{4}$ to 1 ' in diameter. These cocci are to be seen both free and in the blood corpuscles.

With the view of ascertaining how far one or all of these theories apply to the methods of division of the trypanosoma evansi, I have carefully studied all the varieties of forms present in a very large number of stained blood films, taken from the superficial circulation of ponies at different stages of the disease, and from the liver, spleen, and bone marrow of these animals shortly after death. Fresh specimens were also at the same time examined in hanging-drop preparations. Several methods of double staining were used, but it was found that the Nocht-Romanowsky gave the clearest details. The trypanosoma were examined under magnifications varying from 1000 to 3000 diameters. Many drawings were made, and the more interesting are reproduced in Plates V. and VI.

In tracing the generation of the parasite, the first question which presents itself is whether reproduction is preceded by a sexual conjugation. The opinion of the majority of authors is against this theory. The forms described by Plimmer and Bradford as conjugations are regarded by others as phases of longitudinal division. The result of the present observations gives support to the supposition that at times sexual conjugation does occur, and, further, that it is possible to distinguish between the male and female parasite. Three forms of adult trypanosoma can be distinguished by means of the different shape of their posterior end. In some the end is elongated and finishes in a very fine point. In others it is short and truncated, and in the third class it is more or less oval and somewhat resembles the head of a snake. There are reasons for regarding the first class as males, the second as females, and the third as probably females of a younger age. Parasites are frequently to be seen joined end to end with their centrosomes in close proximity and with part of the posterior portion of their bodies closely applied. In many instances the trypanosomata lie almost in a straight line with their ends only fused together. In almost every instance of this kind I have noticed that the parasites are not similar, one being of the first class described, with elongated tapering end, and the other of the second or third class, with short blunt or oval ends.

[1] "Soc. de Biologie" March 1901, and "Annales de l'Institut Pasteur," t. XVI., p. 1.
[2] "Annales de l'Institut Pasteur," t. XVI., p. 241.
[3] "Berliner Tierärztl. Wochenschr.," No. 40, 1903,

It is also a remarkable fact that, almost without exception, the try-
panosomata which appear in various stages of division are those of
the second class. I have not been able to find the forms of the first
class in an undoubted process of division.

In the fresh blood trypanosomata frequently approach each other,
become attached by their posterior ends for one or several minutes,
and break away again.

A close scrutiny will reveal that this is an entirely different process
from any phase of longitudinal division.

It is also not to be confounded with the clumping or agglomeration
of a large number of parasites with their posterior ends in close contact.
This is rather a primary degeneration stage, and is never seen when
the conditions are favourable to the life of the trypanosoma.

After conjugation, in the female the centrosome and the nucleus
divide. As a rule the centrosome divides first, and frequently two
centrosomes and two complete undulating membranes appear before
the division of the nucleus. Reproduction then takes place either by
(1) longitudinal division, or by what may be called (2) transverse
segmentation. In the first method, after the appearance of two cen-
trosomes with attached undulating membranes and of two nuclei, the
protoplasm gradually divides and gives rise to two mature flagellate
parasites. The division may commence from either end, but more
frequently arises from the posterior part. In longitudinal division it
is doubtful if more than two parasites are ever formed from the parent
trypanosoma. I have more than once seen what appeared to be
longitudinal division into three, but the outlines were never sufficiently
clear to preclude the possibility of an accidental deposition of three
adult forms. When reproduction takes place by transverse segmenta-
tion, the body of the parent cell first becomes enlarged and two or
more centrosomes and nuclei appear ; I have frequently seen as many
as four in one parasite. The protoplasm divides into an equal
number of segments, which separate as amœboid forms. These
amœboid bodies develop by stages, in which an undulating membrane
and a flagellum appear and the protoplasm gradually approaches the
mature form, or they again undergo segmentation into a number of
smaller amœboids, which go through a similar process of development
and finally arrive at the mature flagellate organism.

The longitudinal division is a quicker process, takes place in the
blood stream, and results in the direct production of the mature try-
panosoma. The method can be seen at almost any stage of the disease,
and in many fields in stained preparations of blood.

The transverse segmentation forms are more difficult to find. They
can best be observed in films taken at the febrile period and when the
parasites are very numerous in the blood. At the edges of the cover-
glass, and in fields where blood corpuscles are few, they are to be seen
in large numbers.

In the blood at any time the amœboid bodies are few. In the
spleen, liver, and bone marrow, at whatever period of the disease the
animal dies or is destroyed, numerous amœboids are found in various
stages of development. In these organs the adult forms are few.
From this fact it is probable that, after transverse segmentation has
taken place, the amœboids are carried in the blood stream to the liver,
spleen, and bone marrow, and there remain until they develop into the

flagellate parasite. The existence of these two methods of multiplication admits of little doubt. There is also a third and very frequent process of reproduction, the occurrence of which is difficult of direct proof, but the probability of which is supported by many appearances easily observed in the stained films.

In almost every specimen taken from blood, liver, spleen, or bone marrow, and especially in those taken at a time when the parasites are teeming in the blood, very numerous free nuclei can be seen. These take the stain well, and do not appear to be in any process of decay. Some nuclei are naked, while others are surrounded by a slight covering of protoplasm. All stages from the free nuclei up to the amœboid bodies, and from these on to the full grown trypanosoma, can be observed. Frequently trypanosomata can be seen, stained in every detail, with the nucleus at the point of leaving the body of the parasite. It would seem as if these nuclei which leave the body of the living organism, and also those which are freed from the degenerating bodies of dead trypanosomata, possess the power of reforming into a new parasitic element.

DESCRIPTIONS OF PLATES V. AND VI.

(*a*). Drawn with Zeiss apochromatic lens 1·5 mm. Ocular 8 compensating. × 1334 diam. Male and female trypanosomata. Conjugation forms.

(*b*), (*c*), (*d*), (*e*), (*f*), (*g*), (*h*). Drawn with Zeiss apochromatic lens 2·0 mm. Ocular 8 compensating. × 1000 diam.

(*b*). Longitudinal division.

(*c*). Transverse segmentation.

(*d*). Expulsion of nucleus, and reformation into a new parasite.

(*e*). Nucleus leaving body of dead trypanosoma.

(*f*). Development of trypanosoma in liver.

(*g*). Development in bone marrow.

(*h*). Development in spleen.

AFRICAN COAST FEVER.

By WM. ROBERTSON, M.R.C.V.S., Bacteriologist to the Department of Agriculture, Cape Town.

THIS disease is one of great interest and importance, both from the economic and the scientific side ; from the first standpoint in that it will engage the attention of South African legislators in the future more than any other stock epidemic, and from the scientific side inasmuch as research has revealed several deviations from the common type of the class of diseases to which it belongs.

History.—The disease was termed Rhodesian redwater by the first observers in that country. Professor Koch in 1903 gave it the name East African coast fever, and certain authorities in Asia Minor who think the disease exists in that country have given it the name of tropical piroplasmosis. The last is certainly the scientifically correct term, as the disease belongs to the piroplasmosis group, of which redwater (piroplasmosis bigeminum), malignant jaundice of the dog (piroplasmosis canis), and biliary fever in the horse (piroplasmosis equi) are well-known examples.
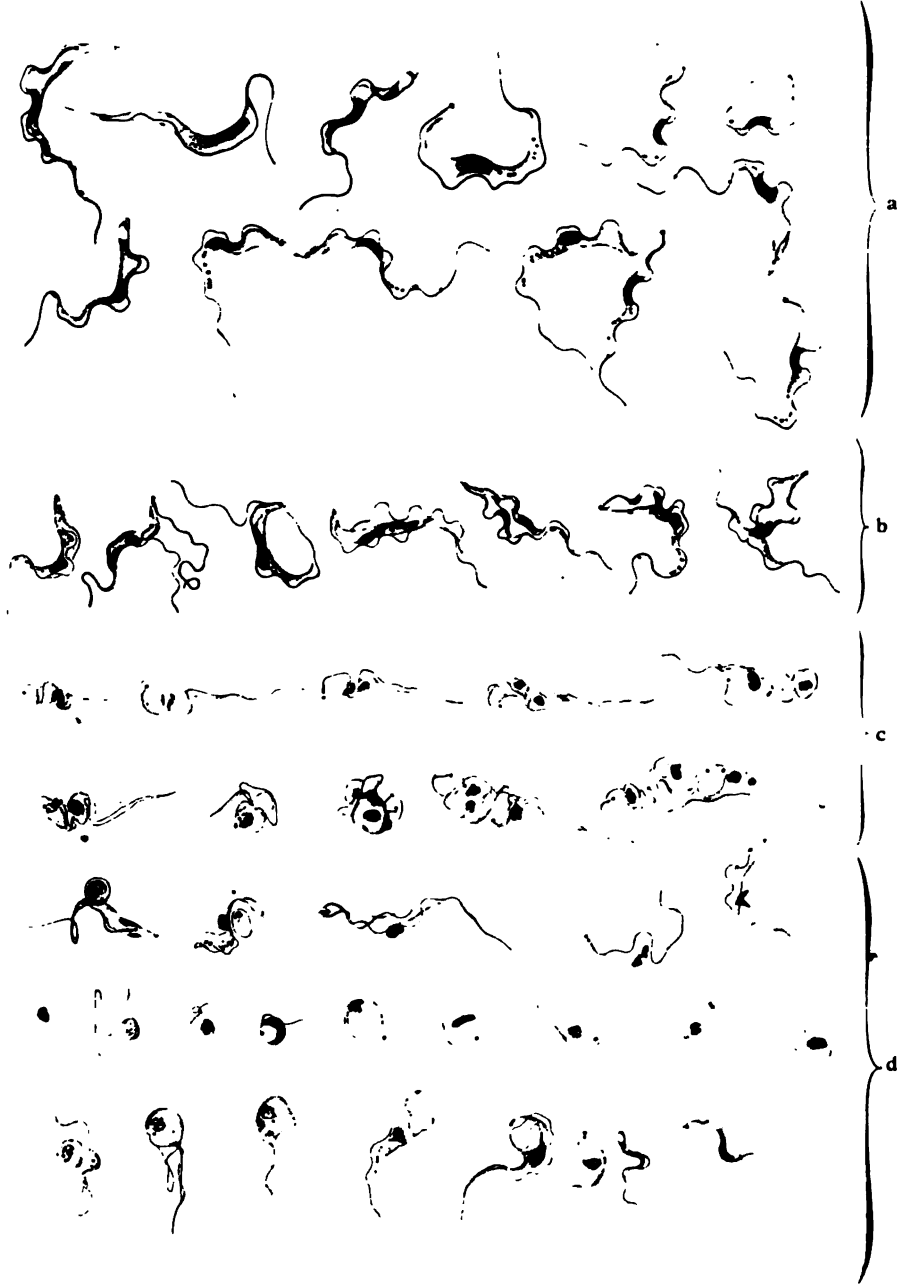
African Coast fever—to give it its present official title—may be defined as a specific disease affecting cattle, characterised by a heavy mortality, high fever, the presence of a protozoon in the red blood corpuscles, and by being not directly transmissible from animal to animal.

Although the mortality amongst horned stock in Rhodesia from this disease did not rise to epidemic proportions until the years 1901-02, localised outbreaks undoubtedly occurred before this, and there is no doubt that of late the gradual infection of the country generally from the Zambezi to Zeerust, and of the transport roads and town lands in particular, had become so far established that all that was necessary to give the disease an impetus was the occurrence of a season which would be particularly favourable for the propagation of that tick which is responsible for the dissemination of the disease.

The first definite notice we have of this disease is in a report by Dr Koch, dated 1897 from Dar es Salem.

The Professor, while working at cattle diseases in that district, described certain intra-corpuscular bodies in the blood of cattle as a modification of the parasite of ordinary redwater—the piroplasma bigeminum. In his report he states :—

"With regard to the forms of the pirosoma and the relations of both it and the full-grown parasite to mild and severe Texas fever, I found in the red blood corpuscles of cases which took a severe form and rapidly proved fatal strange forms resembling miniature staves ; these are frequently curved so as to form rings, and in that case resemble the parasite of tropical malaria."

Redwater itself has been known on the German East African coast and in Rhodesian and Portuguese territory for many years, and the Chief Veterinary Surgeon of Rhodesia states that localised outbreaks of redwater have been known in that territory for the past ten years.

In 1901 a number of Australian (N. S. W.) cattle, about 1000 in number, were landed at Beira as part of the material to re-stock Chartered territory after the Mashona rebellion. These were pastured on Beira commonage, and soon commenced to die from what was considered to be a peculiarly virulent form of redwater. The remainder of the herd were then removed from the coast to Umtali, and from this centre, I think, we can trace the general infection of Northern Rhodesia and eventually Salisbury and Bulawayo.

There is no doubt, looking at the outbreak on Beira flats in the present state of our knowledge, that many of these cattle died from a combined infection of African Coast fever and redwater, and it was the presence of a number of cattle salted to the latter disease which confused the first workers with African Coast fever.

The Transvaal was invaded in 1902, when the disease was noticed at Kommati Poort and Nelspruit, and in November 1902 it was brought to Pretoria by a herd of cattle from German East Africa, which landed at Delagoa Bay, were driven to Kommati Poort, trucked to Machado-dorp, and again driven to Pretoria and sold (sick and dead cattle being left on the road), many being taken to the Rustenberg and Magalies-berg districts.

The last reported infected area was Zeerust (Transvaal).

Nature of the Disease.

It was observed from the first that the disease was not contagious in the strict sense of the word, that certain areas were particularly affected, and that in crossing such areas the cattle began to die from fourteen to fifteen days after.

In the early stages of the disease all work was conducted in an infected area, and, as most of the animals were suffering from combined redwater and African Coast fever, the true lesions of the latter were masked by the former.

It is a well-known fact that an attack of rinderpest will often cause a salted redwater beast to develop clear clinical symptoms of the last disease, and an attack of African Coast fever does the same.

A great number of the cases I saw in Rhodesia presented all the clinical symptoms, temperature charts, and *post-mortem* appearances of Colonial redwater, and the same train of symptoms could be produced in another animal by inoculation.

No doubt these were cases of African Coast fever in salted redwater animals.

Symptoms of African Coast Fever.

The incubative period of the disease is from six to twelve days, and the symptoms during life are not at all diagnostic.

In the early stages of the disease there is a marked elevation of temperature, up to 106° F., with at first comparatively little indication of systemic disturbance. The salivary secretion is slightly in excess, the animal continues to eat, and the bowels are practically normal, though close observation will show that the dung is somewhat glazed in appearance and streaked from an excess in the amount of mucus. The urine generally remains normal throughout the disease.

As the disease progresses the animal appears to be in considerable pain, lies persistently, and rests the chin upon the ground, or on the manger when standing up. The eye is staring and the gait staggering, there is frequently a swelling or œdema of the intermaxillary space, the bowels are loose, and sometimes during the last stages fœtid diarrhœa mixed with blood appears. Not infrequently lung disturbances supervene before the temperature subsides, and the animal manifests great distress in breathing, sometimes coughing like a case of pleuro-pneumonia.

In Rhodesia I saw cases where the disease was prolonged and the lung complications severe, in which extensive subcutaneous emphysema appeared, not as a *post-mortem* appearance, but during the life of the animal, possibly as a secondary consequence of pulmonary emphysema. These subcutaneous swellings crackled upon pressure like those of quarter-evil.

The temperature chart of African Coast fever has the peculiar dip or fall in temperature during the height of the attack which is also seen in cases of redwater (Texas fever).

Post-mortem Appearances.

In describing these, the cases can be divided into those which show lung lesions and those which do not. The following is an example of the latter :—

1. Animal previous to death seemed in considerable pain and had a slight cough ; died rather suddenly. Prescapular, prepectoral, submaxillary, and lymphatic glands at base of tongue enlarged, hæmorrhagic, and œdematous. Tonsils much enlarged. Great congestion of larynx and trachea ; great œdema of lungs, and eleven ounces of clear straw-coloured fluid in the pleural cavity. Kidneys much infarcted with red and white areas. Abomasum much congested, with several ulcers towards the pylorus and œdema of the mucous folds. Spleen normal. Great congestion of the intestinal mucous membrane, and enlargement of the mesenteric lymphatic glands. Great congestion of the cæcum, which contained a large amount of blood in its lumen. Mucous membrane of the colon congested and almost necrotic in patches. Rectum contained mucus and showed spots of congestion on its mucous membrane. Congestion of the pericardium ; petechiæ on the epicardium.

In looking through nearly 200 *post-mortems*, the most constant lesions seen in African Coast fever cases seem to be the œdema and hæmorrhagic condition of the lymphatic glands, the presence of infarcts in the kidneys, and the congestion and ulceration of the fourth stomach. The lung lesions occur in about 30 per cent of cases, the lesions in kidney and glands, so far as my experience goes, in over 90 per cent.

The Causal Parasite.

The parasite of African Coast fever was at one time considered to be an intermediary or immature form in the life cycle of the piroplasma bigeminum. It is now regarded as a distinct species. It is a small piroplasma, and it varies in shape, the two types being the bacillary and the spherical. On staining with selected stain (eosin and azure) the nucleus or karyosome and the cytoplasm can be seen, as in the piroplasma bigeminum. The spherical form resembles an oval or elongated ring, and possesses a staining nucleus. The bacillary form resembles a bacillus, and frequently has an enlargement like a pin's head at one extremity, containing the karyosome.

When we examine the blood of an ox sick with African Coast fever we will, as a rule, be unable to demonstrate parasites in the blood during the first few days of fever ; from then onwards the parasites make their appearance, and they persist and increase in number with every additional day the animal lives. A single red corpuscle may contain from one to five parasites, and both round and bacillary forms are met with in the same cell (*see* fig. 1, Plate VII.).

A curious feature in this disease is the fact that the red blood corpuscles do not decrease in number. In ordinary redwater the normal number of corpuscles (6,000,000 per cmm.) may drop to 1,000,000 per cmm.

Professor Koch has laid down the dictum that the ring-shaped stage in the parasite, when it is met with in an apparently healthy ox, implies that that animal is salted to African Coast fever, and fit for purposes of obtaining blood for his projected inoculation method.

This ring-shaped stage is identical in microscopical appearance with one of the stages of piroplasma bigeminum. Professor Koch had blood smears sent him from Grahamstown, East London, and other redwater areas ; in these blood smears he found a ring-form,

and on this evidence based his statement that African Coast fever existed in Cape Colony.

I imagine my opinion is shared by others—Mr Bowhill, F.R.C.V.S., and Dr Theiler, I know, agree with me—in holding that the ring-shaped parasite seen in salted animals and in cattle from redwater areas is simply a *form of the piroplasma bigeminum*. Mr Bowhill has seen it in the blood of cattle around Grahamstown, which succumbed as a result of attempts to confer an immunity to redwater by injection of salted red-water blood. Mr Dixon, M.R.C.V.S., has found it in smears from the blood of East London cattle, I have found it in cattle which have never been in contact with the contagium of African Coast fever, and "such rings can be produced at any time by injecting the blood of a salted redwater beast into a calf and examining the blood daily." (Theiler.)

If Koch's statement that we have had African Coast fever in this Colony is true, how does it happen that cattle which contain these ring-parasites in them die when subjected to African Coast fever infection, as we know one attack of the latter disease protects?

Again, I have a cow which was born and bred in the Cape, and passed through an attack of African Coast fever, but no ring-parasites are to be found in this animal's blood.

The best stain for demonstrating this parasite is eosin and azure II., the two solutions being mixed at the time of staining. The slides (previously fixed in alcohol and ether) are immersed in this mixture face downwards, in an incubator at 30° F. for two hours. Methylene-blue is not certain, and does not act at all satisfactorily.

Means of Spread and Mode of Infection.

I have found it impossible to transmit pure African Coast fever from animal to animal by inoculation of blood or tissues from an affected beast. Various organs and means of inoculation have all given negative results.

The nature of the disease led observers to the theory that it could be carried by ticks, and Mr Lounsbury, Government entomologist, was the first to prove this experimentally, employing the brown tick (R. appendiculatus) as the agent of infection.

African Coast Fever is carried from animal to animal through the agency of ticks, though probably only by ticks of particular kinds. Mr Lounsbury says—"Two kinds of ticks have thus far been connected with the disease—the brown tick (R. appendiculatus) and the black pitted tick. These two ticks look very much alike, but in the latter species the male is almost black in colour, and has a smoother back, marked with more prominent pits ; the tail is also broader and more blunt. Nearly all the cases of African Coast Fever that have been produced experimentally have been caused by adult brown ticks that as nymphs had fed on cattle in fever with the disease."

A single tick has produced a case.

Mr Lounsbury has conducted a great many tests with larval brown ticks hatched from eggs laid by females off sick cattle, but in no instance has the disease been set up this way.

Similarly, every one of many tests made with young of the very common blue tick (R. decoloratus) have failed. The blue tick stays on the animal during its changes from larvæ to nymph, and from

nymph to adult, not leaving and getting on to a new animal as does the brown tick. It therefore appears certain that the blue tick is not concerned in spreading this disease.

The brown tick fancies bushy situations, is not found on the high veldt, and was in Rhodesia before the disease appeared. The life cycle of the female is as follows: After engorgement the tick drops off the animal, and in a few days lays eggs and dies ; the egg-laying may take weeks, and many thousands may be laid by one tick. The larvæ hatch out in about sixty days, varying with the atmospheric conditions and the temperature.

The hatched larva has six legs, becomes of a brown colour, and crawls to the tops of bushes, etc., to catch the passing host. Having found a host, the larvæ begin to fill themselves with blood, this taking from three to ten days. The engorged larva then drops off, and it is now as big as the head of a common pin. After a fortnight the larva has digested the blood, changed its skin, and become a nymph, which possesses four pairs of legs. Again a host is found, the nymph becomes engorged with blood, and drops off preparatory to a second change of skin and appendages. The nymph is now about the size of a grain of wheat, and resembles a small blue tick. It gets on to an animal, attaching itself by preference to the ears, not deep inside like the red tick, but along the margins.

After mating the female swells very little for a few days, but in a few hours it suddenly swells up to the size of a castor oil bean and drops off.

Mr Lounsbury says the nymphs can live seven months without food. Ticks belonging to the species R. appendiculatus were brought from Rhodesia as larvæ, and produced the disease as nymphs, and others were brought as nymphs and produced the disease as adults. Mr Lounsbury produced a case by transferring an adult tick from a sick to a healthy ox.

Brown ticks taken from the eastern province or farm siding near Cape Town and fed on sick cattle in Rhodesia can produce the disease here after their next moult.

How far the infective tick can pass part of its life history on non-susceptible animals and yet remain virulent for the ox has not yet been demonstrated.

Professor Koch claims to have produced the disease through the agency of the blue tick (R. decoloratus), but, as the Professor's experiments consisted in sowing these ticks on the veldt (and in an infected area), the chances of natural infection were not excluded.

Mr Lounsbury has so far been quite unable to produce the disease except by the brown and black ticks.

Protective Inoculation.

Professor Koch in his third report upon African Coast Fever propounded a scheme of protective inoculation, first by serum and afterwards by salted blood. The first method he abandoned owing to the hæmolytic action of the serum, but the second method was tried on a large scale.

After stating that the blood of salted animals contains the ring-shaped parasites alluded to above, and that the presence of such parasites indicates a suitable animal from which to obtain salted

blood, the Professor goes on to say, " I think that our experiments indicate that the best results may be obtained in protecting against African Coast Fever by using freshly drawn defibrinated blood of recovered animals, and inoculating animals which it is desired to protect subcutaneously with a 10 cc. dose.

" These injections should be repeated four times with an interval of seven days between the injections. Subsequently the inoculations should be continued, lengthening the intervals between the doses to two weeks, and later to a month, the dose remaining the time at 10 cc."

By the courtesy of the Transvaal Government, we received from the Bacteriological Station, Pretoria, two Africander oxen which were said to possess a double immunity, viz., against both ordinary red-water and African Coast Fever, and to have grazed on infected veldt at Nelspruit.

On staining the blood of one of these oxen I was able to demonstrate the ring-shaped, or rather oval-shaped, parasites which Koch said was found in the blood of animals salted to African Coast fever.

Using this blood, I endeavoured to render immune by Professor Koch's method two young Cape cattle. These animals received five doses of 10 cc. subcutaneously, and five doses of 20 cc., also subcutaneously. On being infested with pathogenic ticks they exhibited no resistance whatever, and died from African Coast fever. This form of inoculation has proved quite valueless in practice.

Professor Koch, in addition to this form of inoculation, recommends the slaughter of salted animals, as they are a menace to clean herds. Reasoning on these lines, if one increases the number of salted animals by inoculation one increases the centres of infection, but up to the present the Government Entomologist has not been able to prove that salted animals are a menace to clean herds ; the evidence is all against this theory.

I take it that the most curious and interesting feature of this disease is the fact that it is not directly communicable from animal to animal. I think that amongst the piroplasmoses of animals it is unique in this respect.

Another very interesting fact is the extremely close resemblance between the ring-form of the parasite in African Coast fever and a certain stage in the life cycle of piroplasma bigeminum, for at a certain period they are identical under the microscope.

Preventive Measures.

The first preventive scheme in Rhodesia was prohibiting cattle transport in certain areas and along certain roads, and compulsory dipping of stock.

One or two companies and private individuals in and around Salisbury, Rhodesia, who took up dipping systematically, now have their stock comparatively free from the disease, and have saved a great number of their cattle. They have, I imagine, succeeded in killing off all the infective ticks, and, as the disease, so far as we know, is spread only by ticks, thorough dipping at proper intervals must have a good effect in retarding the disease.

In dipping or using tick-washes it is sufficient if we can find a wash fatal to these parasites. We have stopped searching for one which

will prevent their attacks, and the Government Entomologist is of opinion that a reliable tick - dip must contain arsenic, the non-poisonous preparations living up to their names when used against ticks.

At certain stages of their life cycle, *i.e.*, when moulting, the tick is almost invulnerable to any sort of dip, the double skin acting as a shield and protection.

At the Inter-Colonial Veterinary Conference the following scheme of preventive measures was recommended :—

The disease not as yet existing in Cape Colony, the Conference recommended that measures for the eradication of African Coast fever be taken only by the Transvaal in their affected districts and in matters of boundry fences, the other Governments of South Africa to contribute towards the expense.

All affected cattle to be slaughtered without compensation, and the disinfection of hides and carcases to be carried out.

In contact animals to be dipped and moved, or, at the discretion of the veterinary officer, slaughtered, and compensation given at a fixed tariff; place of slaughter to be also at the discretion of the veterinary surgeon who supervises disinfection, etc.

All infected areas to be cleared of cattle for eighteen months, and any cattle straying into such areas to be immediately destroyed without compensation.

Burning of the grass was strongly recommended.

It was estimated that the number of cattle destroyed in carrying out this scheme would not exceed 10,000, and the probable cost £50,000, but this will not include the cost of veterinary and police supervision.

The Transvaal, Natal, and Swaziland border to be fenced (cost estimated at £10,000), and a one mile police patrol belt to be instituted.

The cost of carrying out these plans to be borne by the various Colonies, and its distribution based on the number of horned cattle in each State as given in the recent census returns.

JAGZIEKTE OR CHRONIC CATARRHAL PNEUMONIA (SHEEP).

By WM. ROBERTSON, M.R.C.V.S., Bacteriologist to the Department of Agriculture, Cape Town.

THERE is a disease affecting the lungs of sheep during the winter months in the upland parts of this Colony to which the Chief Veterinary Surgeon has given the name of chronic catarrhal pneumonia, and which is known locally as "jagziekte."

It accounts for a considerable number of animals every year, and from its characteristic lesions I am inclined to think it is due to some specific cause other than exposure to cold and wet.

It is a peculiar form of pneumonia, inasmuch as the hardening and consolidation of the lungs described below is not preceded by the

usual stages of congestion, hepatisation, etc., and there is an almost
entire absence of fever.

Symptoms.—The first symptom usually noticed is a cough, with
marked shortness in depth and increase in number of respirations.
These symptoms as the disease progresses simply increase in severity,
until the least movement causes the animal to pant for breath, heave
at the flanks, and lie down completely exhausted.

It is this heaving of the flanks and hurried breathing which gave
rise to the Colonial name of "jagziekte," from "jagt," the Dutch to
drive, and "ziekte," a sickness.   The animal looks as if it had been
over-driven, and that is quite the impression a casual observer might
get if he saw two or three affected sheep trailing after the flock.
Another Colonial name is "hartslagziekte," the "hartslag" being a
general name for the heart, lungs, and liver, *i.e.*, the pluck.

The course of the disease is more rapid in winter than in summer,
and careful treatment and nursing undoubtedly prolong the animal's
life.

I have had an animal under observation in a shed for seven months
which, though slightly affected when brought in, gradually grew worse
and worse, until it undoubtedly died from want of lung area.

Treatment.—The Chief Veterinary Surgeon shares the opinion as to
its contagious nature, and recommends the slaughter of all affected
animals.

Post-mortem Appearances.—It is not easy to obtain good, that is,
advanced *post-mortems*, as the sheep-farmer usually slaughters a sheep
whenever anything is noticed wrong with the breathing and uses the
flesh, but the following are the *post-mortem* appearances in a moderate
case of the disease.

The lungs and their attendant lymphatic glands are alone affected ;
the latter are enlarged, pale, and œdematous.   The lungs show more
or less extensive areas of consolidation ; sometimes there seems one
centre or area of infection, but other cases seem to indicate that the in-
fection has started from many centres.   The affected part is quite solid,
of a darker colour than normal lung, firm, friable, and slippery to the
touch.   In some cases the affected part cuts like bacon, and the cut
surface has a soapy, greasy, feeling to the fingers.   In advanced cases
fibrous tissue may form to such an extent that the affected part of the
lung cuts like cartilage.   The bronchi persist, but the lumen is often
occluded.   The consolidated portions do not appear either congested
or swollen.   On squeezing the cut surface a quantity of froth and *débris*
oozes from bronchi.   The plugged air cells do not empty when scraped.
Sometimes on section the plugged bronchi stand out like tubercles, and
the lungs may be as dry as cartilage.

In typical cases of this disease there does not appear to be any
tendency towards breaking down of the lung tissue or formation of
abscesses.

"The pleura is not affected in the early stage, but after the disease
has extended so as to involve a considerable portion of the lung the
pleura over the affected part becomes thickened, and inflammatory
adhesions form between the lung covering and the lining of the ribs.
The great majority of the affected animals die, though many linger on
for months if allowed to do so " (D. Hutcheon).

One thing which strikes an observer at a *post-mortem* is the sharp

line of demarcation between comparatively healthy and completely consolidated lung. This is seen in Fig. 3 (Plate VII.) ; here the almost occluded air cells and bronchi approximate in the same field to what is only slightly affected tissue.

Microscopical Examination.—The lesion as shown under a low power (Figs. 2 and 3, Plate VII.) is that of catarrhal pneumonia. The alveoli are plugged with an exudate which consists almost entirely of proliferated cells from the alveolar lining, mixed with a few leucocytes. The affection appears to spread steadily from sick to healthy lung.

Composite drawing from three fields, showing crescent-shaped bodies (Leitz ₁₂ oil imm., No. 4 comp. ocular).

While examining smears from affected lungs I have noticed the presence of a body which I take to be of a parasitic nature. I am unable to find it in healthy lungs or in healthy parts of lungs which show areas of the disease. This parasite is a crescent-shaped body with rounded ends, about twice the diameter of a red blood corpuscle in length, with a centre which takes the stain deeply in the form of a band, and in certain particulars recalls the crescentic forms of human malaria (Laverans bodies).

Another body which I take to be a different stage of the same para-

site is a small cylinder with rounded ends, resembling a segment of a ruler.

These parasites are rather sparing in number, and very difficult to stain. For some time 1 was attracted by what I thought were vacuoles or grease globules; these were the parasites which did not take the stain.

I stain with warm carbol-fuchsin for twenty minutes, then soak in weak spirit and water.

The bodies I describe, though I do not claim them to be the causal parasite of the disease, are of sufficient interest to warrant a description. I take them to belong to the sporozoa, members of which group are credited with pathogenic characters.

Rixford and Gilchrist describe in detail two cases of Protozoon infection of the skin (John Hopkin's Hospital Report, Vol. I., page 209, 1896), and the bodies seen in this chronic cattarrhal pneumonia may be the cause of the lesions just described.

In conclusion, I may state that up to the present I have failed completely to convey the disease from sick to healthy animals by cohabitation, feeding, and all varieties of inoculation methods, both with blood and affected tissues.

This disease I think is mainly worth note because of its insidious progress and incurability, and because it shows no tendency for the affected parts of the lung to undergo degeneration, caseation, or the formation of abscess.

DESCRIPTION OF PLATE VII.

African Coast Fever.

FIG. 1. Exact reproduction of a microscopic field in a blood film from an advanced case of African Coast fever. Stain azure II. and eosin. Zeiss $\frac{1}{12}$ oil imm., ocular 4 (*see* p. 217).

Jagziekte.

FIG. 2. Section of badly affected lung, showing complete blocking of the alveoli with proliferated epithelium.

FIG. 3. Section showing junction of badly affected and comparatively healthy lung tissue.

IMMUNITY.[1]

By WILLIAM BULLOCH, M.D., Bacteriologist to the London Hospital, E.

THE advent of the bacteriological era in medicine twenty-five years ago revived the old question whether resistance to the invasion of infectious virus is brought about by the body humours or the body cells, and just as the old humoral doctrines preceded Virchow and his cellular pathology, so here the humoral doctrines are chronologically the first. In dealing with the question of the relation of the cells and fluids to the immunity problem—a problem which for interest to the
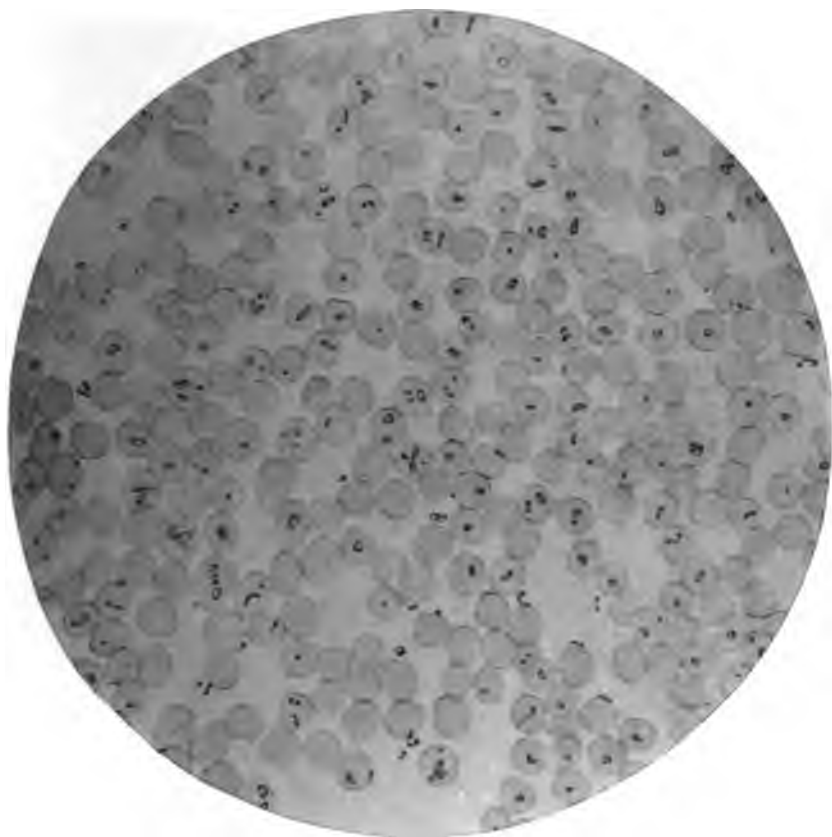
Fig. 1.

Fig. 2.
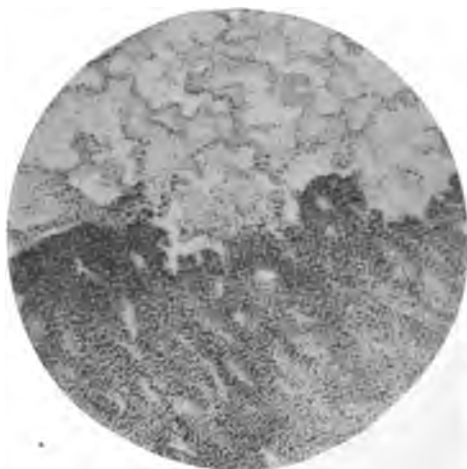
Fig. 3.
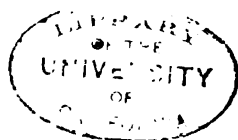
medical scientist is without its rival at the present time—it seems to me necessary to pause and consider the foundations of our beliefs as progress towards the final goal—the construction of a rational system of therapy based on the etiological curative principle—can only be obtained by following the great currents and by avoiding the eddies and backwaters which lead nowhere. In the present instance I have attempted by a study of the extensive literature and by independent observation to determine what is fact and what is fiction in regard to the question of the cellular pathology and immunity. Too often even the youngest of us have seen beliefs taught to us as facts swept away because their foundations were insecure, and isolated experiments on animals of lowly organisation have been utilised without further criticism for the construction of hypotheses applicable to problems of vast complexity in the higher mammalia. The Pathological Committee of this Association has, I think, done wisely to discuss at the present time the doctrine of immunity in its various aspects, so that by observation, criticism, and study we may direct our course into paths likely to be heuristic, and that we may distinguish incontrovertible facts from what is doubtful, and from what perhaps may better be referred to the domain of myths.

That there is a condition of immunity which can be artificially produced is certain, and we owe the basis of this knowledge to the classical work of Pasteur on *Cholera des Poules*, Pasteur and Thuillier on *Rouget*, and Pasteur, Roux, and Chamberland on *Anthrax*. Not satisfied with the mere existence of immunity, attempts were then made to solve the nature of the immunity and its mode of action. Certain facts were observed, and then speculations arose as to how these facts are to be interpreted, and from this time dates the difficulties which have arisen in the immunity question. From actual observations in flasks of broth with cultivations of the chicken cholera bacillus, Pasteur formulated his *theorie d'épuissement*, or exhaustion theory, to explain what takes place in the body of the fowl immunised by the hand of man. Now, it often happens that a theory, although it contains a great element of truth, may be subverted, because it cannot at the time satisfactorily explain particular observations. Such has been the fate of Pasteur's theory because it could not explain the fact that immunity can be produced by the soluble products of microbes, and because the tissues of animals, immune or otherwise, suffice perfectly as a medium for the growth of microbes. (Bitter, Griffin, Metchnikoff.) Although, therefore, Pasteur's theory cannot be held to explain the whole question of immunity, it is still a question whether certain forms of natural immunity may not owe their existence to an absence of suitable nutritive pabulum—a view revived in recent years by Ehrlich in the conception of the absence of suitable receptors.

Chauveau on the analogy of the noxious substances produced by microbes in artificial media, thought that in the living body substances might be retained, these acting detrimentally on the microbe, and this was the foundation of his doctrine or *theorie de la substance ajoutée*. In his belief he was supported by the fact that the lambs of sheep vaccinated against anthrax may be born immune; and secondly, that it is possible to immunise by means of soluble products of bacteria.

Both these old theories neglect what is now known to be a funda-

P

mental factor, namely, the host infected and the means employed by that host to resist microbic infection. This was first clearly set forth by Metchnikoff, whose writings have played an immense rôle in the elucidation of this whole problem. As is well known, Metchnikoff's theory of immunity is that the cells bring about the resistance to infection, and this is based upon a number of observations, notably the discovery by Haeckel (1862) that amœbæ englobe foreign bodies, the discovery of Langhans (1870) that emigrated leucocytes in hæmorrhagic foci became charged with red blood corpuscles. Birch-Hirschfeld (1872) had found that micrococci injected into the blood are taken up by leucocytes, and Panum had suggested that possibly this was a method by which microbes might be destroyed.

Röser expressed himself much more definitely that the immunity is due to the ability of contractile cells to seize the infectious virus.

Long before this Metchnikoff (1865) had himself shown that the intestinal protoplasm of Geodesmus, a land planarian, is capable of englobing foreign bodies, and from about 1878 onwards he has in a long series of researches laid the foundations of his so-called phagocytic theory. These foundations cover a wide zoological field, ranging from the lowly rhizopoda to the highest mammals (Metchnikoff). Briefly, the theory held by Metchnikoff was that the virus was destroyed in the interior of certain mesodermic cells by a process of digestion. In recent years Metchnikoff has had to shift his standpoint to a certain extent, although in the main the cardinal doctrine is the intracellular digestion in leucocytes and other cells of mesodermic or even epiblastic origin.

While Metchnikoff was collecting his data and building up his hypothesis another theory was gradually being evolved—the humoral theory defended chiefly by Buchner, and at first this was directly antagonistic to the views promulgated by Metchnikoff.

The humoral theory was based on certain experiments by Traube and Geschleiden, Wyssokowitch, and Fodor, who observed that bacteria injected into animals cannot be recovered again although the same bacteria did not appear to have made their exit from the body.

From 1888 dates a long series of researches inaugurated by the important memoir of Nuttall, showing that destruction of bacteria takes place in cell-free serum outside the living body altogether, and that the bacterium-destroying property in the serum can be inhibited if the serum be heated to 56° C. This fundamental experiment has been confirmed by numerous workers, among whom one may mention Behring, Nissen, Lubarsch, Stern, Denys and Kaisin, de Giaxa and Guarnieri, Leclef, Denys, Buchner. In particular Buchner has elaborated this branch of work in a long series of elaborate memoirs, culminating ultimately in his alexic theory of immunity. In spite of various objections raised by Christmas, Székely and Szana, Baumgarten and his pupils, it is now accepted as proved that in the serum there are substances—alexins—which destroy certain bacteria *in vitro*. Buchner has drawn special attention to the characters of the alexins —their thermolability, their absorption by bacteria, and their behaviour on dialysis.

Leaving the question of the existence of alexins, we find ourselves

on very uncertain ground when we come to the problem of the site and the mode in which Buchner's alexin is manufactured in the body.

Hankin was the first to refer the alexins to a leucocytic source, but erred in regarding the eosinophilic cells as the alexocytes (Mesnil), There is no doubt, however, that his work led to other researches. which culminated in a partial fusion of the rival humoral and cellular theories. These researches are closely connected with the names of Denys and his collaborators in the Louvain School, and were accepted by Buchner in his address at the Congress for Hygiene and Demography in Buda-Pesth in 1894. The opinions of Denys were further strengthened by the observed correlation between hyperleucocytosis and alexic action (Sanarelli, Everard, Massart and Demoor, and Bordet).

Before dealing with this important question of the connection of the leucocytes to the amount of alexin, it will be remembered that in point of time the discovery of antitoxin had insinuated itself into the immunity problem, and the same humoral and cellular difficulties again arose. Is this remarkable substance antitoxin merely in the humours or is it a vital cell product? That the latter view is correct is apparent from the researches of Roux and Vaillard, Salamonsen and Madsen, Knorr, and constitute a support for Metchnikoff's views of the cellular origin of antibodies, the whole question being, Are we, or are we not, in a position to locate this cell factory? Is it limited to one type of tissue, as affirmed by Metchnikoff, or not? In all his publications Metchnikoff has definitely committed himself, and has asserted the pre-eminent rôle of the leucocyte, and most of the researches emanating from the Pasteur Institute have been dominated by Metchnikoff's ideas.

We have already referred to the possible origin of alexins from leucocytes, and we must now consider carefully the grounds on which Buchner accepted this new view as expressed by him at Buda-Pesth in 1894, and why he departed from the purely humoral conception, for he definitely stated that the leucocytes possess important functions as defensive organs, and especially from their ability to secrete alexins. Phagocystosis he regarded as a secondary phenomenon.

Denys and Havet found that dog's serum is less bactericidal than dog's blood, and special attention was directed to the factors to which this could be due. They tried blood which had been filtered through paper, and serum with and without the addition of leucocytes, and using as test objects bacterium coli, bacillus subtilis, and staphylococcus, they came to the conclusion that by filtration dog's blood loses practically all its bactericidal properties, and as the leucocytes were kept back by the act of filtration, they assumed that they must in some way be connected with the formation of alexin. The blood of man, pigeon, or fowl did not, however, yield similar results. The microscopic examination permitted the observation of all stages of phagocytosis, and although they thought that the serum itself plays a very small part in bactericidal action, they stated in their conclusions that neither the phagocytic nor the humoral theories taken separately are sufficient to explain immunity, but the cells and fluids act together in a manner varying with the species of animal, and probably also with the nature of the virus.

Havet observed an increase of bactericidal power following a leucocytosis produced by injections of staphylococcus cultures.

By means of exudates artificially produced by aleuron emulsions, Buchner found that the leucocytic fluids were more bactericidal on bacillus coli than serum or blood. The question of phagocytosis was put out of court by killing the leucocytes by freezing and thawing. Denys also believed that the alexins were secretory products of the leucocytes. The experiments of Buchner were repeated with variations, who came to similar conclusions. In order to obtain leucocytes as isolated as possible, he introduced into the peritoneal cavity small pledgets of sterile cotton-wool, and after a number of hours these were removed and squeezed, and the fluid frozen and thawed, and then tested. The results were not quite harmonious, for although the bactericidal action of the leucocytic fluid for bacillus typhi and staphylococcus was greater than that of the serum, the contrary was found to be the case for vibrio choleræ. From Hahn's calculations it is not apparent that the bactericidal action on staphylococcus was very pronounced, or even beyond the limits of experimental error. Schattenfroh made an extensive series of observations with leucocytes more isolated than had been the case in the experiments of Denys, Buchner, Hahn. The leucocytes were thoroughly washed in saline solution, and then centrifugalized. They were then added to the inactivated serum, and the bactericidal action was observed.

Schattenfroh also found that if isolated leucocytes were frozen and then mixed with inactive exudate and macerated for one to two days in the cold they acted bactericidally on certain bacteria.

From this period onwards a very large number of researches have been published to determine the question of the exact relationship of the leucocytes to the alexins, and as time has gone on it has become apparent that the question is a complicated one. This is the outcome of investigations by Bordet, Ehrlich and Morgenroth on hæmolytic serums and from the fact that Buchner's alexin is a complex of two distinct substances, namely, a complement (alexin proper) and an immune body (amboceptor. Ehrlich).

As is now well known, the injection of alien erythrocytes leads to the development of a hæmolytic serum, the hæmolysis being due to the co-operation of a thermolabile complement—also called alexin and thermostable immune body, otherwise amboceptor.

Clinging fast to his cellular theories, Metchnikoff believes that both these bodies are contained in certain cells or at any rate are produced by them, and that in the main cells like erythrocytes are destroyed by certain ferments—cytases—present in the macrophages. The existence of these cytases is based on experiments with protozoa and actinia. Experiments with neutral red, led Mouton to the belief that amœbæ possess a ferment—amibodia stase. Similar ferment, actinodiastase, was extracted from actinia by Mesnil. Arguing from this Metchnikoff supposes that the leucocytes are similar and that they also possess endocellular cytases.

For a number of years Metchnikoff has distinguished two great types of phagocytes, namely, macrophages and microphages, the former possessing no granulations, the latter including the neutrophile and eosinophile leucocytes. The exact histological characters

of the macrophage are to my mind not apparent from Metchnikoff's writings. He sometimes describes them as large lymphocytes and speaks of their chief site as being the lymph glands, epiploon, and spleen. At other times (p. 84) he says that the mesoblastic phagocytes are divisible into fixed phagocytes or macrophages of the spleen, endothelium, connective tissue, neuroglia, muscular fibres, and into free phagocytes. He also tries to show that the macrophages and microphages act differently, in so far that the macrocytase destroys red corpuscles and cells, the microcytase being chiefly concerned with microbicidal action, and he parallels this conception with the existence of other leucocytic ferments, such as fibrin ferment, amylase, lipase, and oxidase.

Proceeding still further, Metchnikoff regards the identity of the cytases with Buchner's alexin as demonstrated, and that the alexic action is of the nature of a digestive process, although this is entirely negatived by the experiments of Nolf, Gruber, and others. He also assumes that any macrocytase which is present in the serum has come there by the phagolysis of the macrophages. The discovery of the complex character of cytolytic serums no doubt was a puzzle to Metchnikoff at first, but he rose once more to the occasion by affirming that the amboceptor, or, as he prefers to call it, the fixator, is also phagocytic in origin, and is produced in abundance during intracellular digestion. When it is produced in excess it escapes from the macrophages, and makes its way into the serum, in which it can be demonstrated in large quantity.

We have in these statements the most concrete and most extreme views yet published on the relation of cells to immunity, and it now behoves us to consider whether these views have met with the support of other investigators.

I have already dealt with the views of Denys, Havet, Hahn, and Schattenfroh on the alexic secretory action of leucocytes, and we have to discuss whether there are real grounds for this belief.

In the first place, it may appear strange that the simple question whether the alexin is free in the plasma or not has been the subject of numerous researches of contradictory character.

Metchnikoff, basing his conclusions on the work of Gengou, affirms that the plasma is alexin-free. Gengou examined the blood or rabbits, dogs, and rats in paraffined vessels to prevent coagulation and failed to demonstrate bactericidal action in the plasma for bacillus typhi, bacillus anthracis, bacillus coli, and V. Metchnikovi, a result confirmed by Levaditi ; Petterson, on the other hand, using oxalate and citrate plasma, got entirely different results in the case of bacillus typhi and bacillus coli. Von Dungern found that the plasma of scyllium canicula was quite as hæmolytic for rabbit's corpuscles as the serum. Hahn could find no difference between the serum and histon blood. Similar results led Simnitzky and Lambotte to the belief that alexin or complement circulates free in the plasma.

The Munich school, influenced by the work of Buchner, Hahn, and Schattenfroh, and regarding the alexin as a secretory product of the leucocytes, have attempted by a variety of methods to extract alexin from these cells. Van de Velde used distilled water as an extractive. Bail used leucocidin, Löwit ground up the leucocytes with glass powder. As a result of numerous experiments Schattenfroh came to

the conclusion that the substances which can be extracted from the leucocytes are not identical with the serum alexins. Shibayama examined the effects of extracts of guinea-pig's organs on dog's blood and found that hæmolysis was produced by splenic and lymphatic gland extracts, but not by bone marrow or other organs. Tarassewitch examined the hæmolytic action of extracts of organs of guinea-pigs, rabbits, and dogs, and found the so-called macrocytase in the lymph glands and other macrophagic organs, a conclusion also supported by the experiments of Levaditi. Korschun and Morgenroth found that certain organ extracts were hæmolytic, but that the hæmolytic substances present were quite different from alexins, as they were very heat-resistant, soluble in alcohol, and incapable of forming antihæmolysins. Similar results were obtained by Donath and Landsteiner and Doemeny.

Petrie was unable to find any bactericidal substance for bacillus typhi in any of the leucocytes either of the normal or the immune rabbit. Nor was he able to reactivate an inactivated serum by the addition of leucocytes or leucocytic extracts. Similar negative results were obtained by Ascher in the case of vibrio choleræ. In a study of the source of hæmolytic complement and amboceptor, Bulloch, by simultaneously measuring the amount of the hæmolytic constituents on the one hand and the mononuclear and polynuclear leucocytes on the other, found that the appearance of the amboceptor in the serum is coincident with a rise in the number of mononuclear leucocytes, and the latter follow the fluctuations of the former with regularity.

By producing an artificial polynuclear pseudo-eosinophilia there is a coincident rise in the amount of the complement, but no rise in the quantity of immune body. Ainley Walker also believes that complement is a leucocytic product appearing in the blood plasma mainly as a result of disintegration of the leucocytes. Studying the serum complement in disease, Longcope observed a rise in the quantity of complement associated with hyperleucocytosis.

In the case of immune serums, Pfeiffer and Marx, Wassermann, and Deutsch found the main site of formation of antibodies to be the spleen, bone marrow, and lymph glands.

Taking advantage of the fact that a certain time—usually four or five days—elapses between the injection of ox blood and the development of the specific hæmolysis in the blood of rabbits, I attempted, by making extracts from the different organs, to determine where the hæmolysins were formed ; but the technique was so difficult and the results so uncertain that the research was not continued, but so far as it went there was no evidence that more hæmolysin was formed in the spleen and bone marrow than in other organs.

On the other hand, Wauters, experimenting with staphylococcus and hay bacillus in the case of pigeons and rabbits, and making extractions of the organs with inactivated serum, found the most marked bactericidal properties in bone-marrow extracts. Briscoe and Moxter could find no immediate relation between the microphages and the presence of bactericidal complement.

Other researchers are not only satisfied that this relationship exists, but they have sought to demonstrate the manner in which the leucocytes part with their alexin. Laschtschenko used as a leucocytic extractive a serum heated to 55° C., and not in itself bactericidal, and

he assumed that in such a serum the leucocytes were living, and that the increased amount of alexin must necessarily be due to a secretory activity on the part of the leucocytes. Trommsdorf, repeating this work, estimated the vitality of the leucocytes by their amœboid movement and by their staining reactions with Nakanishi's method, and he found that the majority of the leucocytes were living. Most recently Lazar, doubtful as to whether these estimates of vitality were sufficient, took as his test the ability of the leucocytes to exert a phagocytic action, and he ultimately came to the conclusion that bactericidal substances get into the serum only when a certain number of the leucocytes are destroyed.

From this mass of conflicting details it would appear difficult to draw accurate conclusions in regard to the origin of protective substances. In the first place, the existence of alexins is undoubted, but nothing certain is known of their origin. Even the question of their existence in the plasma is not definitely settled, although the balance of opinion is in favour of their concurrence outside the blood cells. It is not certain that cytases exist in Metchnikoff's sense. It is very improbable that alexin acts like a digestive ferment. It is not proved that leucocytes secrete alexin, although it is probable that they do.

It may be asked why there is this extraordinary diversity of opinion, and the answer must be sought in the complexity of the subject and the defects in the technique employed, for there is no doubt that the experimental error in determining the number of bacteria destroyed, especially when this estimation is carried out by the older plate method, is a very great one. This is clearly seen when one comes to an organism like staphylococcus—an organism much in favour in this class of work. According to the best bactericidal methods of Wright, it is impossible by the aid of a serum, normal or immune, to kill a staphylococcus by bacteriolysis, using the term in its technical sense.

The result of all this investigation has been the development of a great harmony between the views of the rival schools of Buchner, Ehrlich, and Metchnikoff, and it would seem at the present time that the differences are more those of words than of actualities. Perhaps the main point still is that the French school attribute to the leucocytes a more important rôle than that admitted by the Germans, and there is a great amount of evidence that the leucocytes are of importance, although the exact manner of their action is still unsolved. We have spoken of antitoxic immunity and bactericidal immunity, and we may ask ourselves whether the host has exhausted its resources against the virus or the poison by these two methods. In other words, is immunity always antitoxic or bactericidal? and I use the word "bactericidal" in a specific sense and not in its etymological sense. According to Nuttal, Stern, Neisser, and Wright the serum of an animal is not bactericidal towards staphylococcus. I have found the same in the case of B. pyocyaneus. No doubt staphylococcus and B. pyocyaneus are destroyed in the living body, but not by the specific method we know as bacteriolysis. Now it is a remarkable thing that for the majority of infective diseases the immunity is neither an antitoxic nor a bactericidal, and it is chiefly in the case of these diseases that Metchnikoff's phagocytic theory was established, and he has always clung to the belief that the serum

plays a subordinate rôle compared with that of the cells. By a study of the serum and the phagocytes separately Wright and Douglas, in two epoch-making papers, have shown that in so-called phagocytosis a most important, if not a cardinal, rôle is played by substances in the serum, which substances have nothing to do with Buchner's alexins. Their method has been to mix serum or plasma with bacterial emulsions and washed leucocytes, and after contact of these for fifteen minutes films are prepared and stained by Leishman's dye, and the number of bacteria ingested by the leucocytes is taken as the index of the phagocytosis. This method is a modification of that originally brought forward by Major Leishman in his studies on phagocytosis.

Wright and Douglas found that the phagocytic property is arrested by heating the serum to 60°-65° C. for ten minutes, although the leucocytes are, of course, normal ; that is to say, the leucocytes are practically unable to pick up microbes by themselves. If the unheated serum is mixed with bacteria at 37° C. for fifteen minutes and the mixture is then heated to 60° C. for fifteen minutes, phagocytosis can still take place, thus demonstrating that the serum acts in some way on the bacteria, rendering them suitable prey for the phagocytes. This thermolabile serum feast preparer is called by Wright and Douglas opsonine (ὀψωνιαζω, to furnish with provisions).

They have also shown that during the process of active immunisation the opsonic value of the serum is increased, and they have succeeded in demonstrating this opsonic immunity for a number of infections, such as staphylococcus, Malta fever, pneumococcus, tubercle. In conjunction with E. E. Atkin I have devoted considerable time during the last six months to the question of opsonic immunity, and we have been able to confirm the work of Wright and Douglas, and have studied in other directions the mode of formation and action of opsonic serum. We have found it impossible to increase indefinitely the amount of opsonine in the serum as a result of inoculation. A limit is apparently reached very soon, so that if the serum is diluted the opsonic effect is not manifest. The effect of injections of microbes on the opsonic content of the serum is similar to the effect on the antitoxic or bactericidal content. There is, as is well known, a negative phase, a positive phase, and a higher base level. The reason why the opsonic value cannot be indefinitely increased by inoculation is difficult to understand, especially as we have found that the opsonine is a body built on the type of agglutinin, and analogous to Ehrlich's receptor of the second class. Opsonic serum which has been heated cannot be activated again, although this inactive opsonin is seized by bacteria and fixed to them, so that when mixed with fresh active opsonin no opsonic effect is apparent. We must admit, therefore, the existence of an opsonophoric and a haptophoric group. Further, we have demonstrated that the serum contains a number of opsonines, each of which is specific for given bacteria. By saturation experiments we can remove the one opsonine after the other, our results being in harmony with the pleurimistic doctrines of Ehrlich. From the researches of Wright and Douglas, Bulloch and Atkin, it would appear that in addition to antitoxic and bactericidal immunity there is a third type, namely, opsonic immunity, which is a common phenomenon, and is brought about by the coalition both of humours

and cells, and it corresponds largely to what Metchnikoff has preached for years as phagocytosis. The actual part played by the leucocyte in the drama seems less clear than ever, as evidently the bacteria are altered in some way before being ingested. This alteration is not, however, a fatal one to the microbe, and no doubt plenty has to be done by the leucocyte before the microbes are disintegrated.

Thousands of facts point to the conclusion that our leucocytic-forming tissues are our great defensive organs against parasitic invasions. The mystery is how the microbes are destroyed, and in this connection we seem pretty much in the same state as John Hunter over a century ago, when he wrote of pus: " The final intention of this secretion of matter is, I believe, not yet understood, although almost everyone thinks himself able to assign one, and various are the uses attributed to it."

EDITORIAL ARTICLES.

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THE TREATMENT AND PREVENTION OF SHEEP-SCAB.

In the month of April 1903 the late Mr Hanbury, then President of the Board of Agriculture, appointed a Committee " to investigate experimentally and to enquire into and report upon :—

"(1) The composition and essential constituents of efficient dips and other preparations for the treatment and dressing of sheep, and their effect upon the animal treated or dressed and upon the parasites and other organisms for the destruction of which they are used ;

"(2) The methods in which such dips and other preparations should be employed, and the appliances and facilities requisite for the purpose ;

"(3) The times and intervals at which sheep should be treated or dressed, regard being had (a) to the life-history and characteristics of the sheep-scab acarus and of the other parasites and organisms of sheep which require external treatment; and (b) to the practical conditions under which sheep-farming is carried on in various parts of the United Kingdom."

The Committee's Report and the Minutes of Evidence taken by them have recently been published, and the principal recommendations contained in the former are given at a later part of this number. Although the Minutes under which the Committee was appointed were so drawn as to cover the treatment necessary to combat all the common external parasites of the sheep, the enquiry was mainly

directed to the subject of sheep-scab ; and, in brief, the findings of the Committee amount to an expression of opinion that the only method by which that disease can be eradicated is compulsory annual dipping of the whole of the sheep in the country, and a recommendation that the Board of Agriculture should exercise the powers conferred on them by the Diseases of Animals Act, 1903. This was in reality a Sheep-scab Act, extending the powers conferred by the Diseases of Animals Act, 1894, in two important respects—viz., (1) by empowering inspectors of the Board of Agriculture or of a Local Authority to enter any premises and examine any sheep therein ; and (2) by authorising the Board of Agriculture to enforce "the periodical treatment of all sheep by effective dipping."

No one who remembers that the sheep-stock of the British Islands amounts to nearly thirty million head can fail to appreciate the magnitude of the task which the Committee are anxious to see undertaken, or deny that the attempt to enforce simultaneous annual dipping of all the sheep in the country should not be begun unless there is forthcoming sufficient evidence to show (1) that such dipping is really practicable, having regard to the conditions under which sheep-farming is carried on in various parts of the kingdom ; (2) that such dipping would be effective for the purpose in view—viz., the eradication of sheep-scab ; and (3) that the same end could not be secured by other and simpler methods. We therefore think it well to devote some space here to an examination of these important questions.

Let us consider in the first place the question of the practicability of the scheme which is propounded. Stated with the omission of all details, the scheme is one to ensure that at a certain season of the year every sheep in the country shall be immersed in one or other of a series of dips approved for that purpose by the Board of Agriculture. In referring to the scheme above, we have used the expression " simultaneous annual dipping," and we find that the word "simultaneous" was sometimes employed by members of the Committee in eliciting the opinions of witnesses as to the feasibility or advisability of compulsory general dipping. In reality, however, what is meant is the dipping of all the sheep in the country between two specified dates within six months after shearing. As to what ought to be the actual length of this dipping period, the Committee have not expressed any opinion in their report ; but from the minutes of evidence it may be gathered that it would probably be one or two months, or, say, August and September. In considering whether it would be possible to ensure that every sheep should be dipped once within this period, it must be remembered that the operation would be useless for the purpose of curing or preventing sheep-scab unless it were carried out in such a way as to secure a certain period of complete immersion in one of the specified baths. Everyone who has had anything to do with super-

vising the execution of prophylactic measures by stock-owners will recognise that it would be folly to expect that this compulsory dipping would be properly carried out without what may be called legal supervision. This vital question of supervision has not been dealt with by the Committee in the way that it deserved. It is true that in one paragraph they recommend the appointment " of one or more veterinary or other inspectors for a county or a group of counties," for the purpose of seeing to the due execution and enforcement by the Local Authorities of the Diseases of Animals Acts, including any orders relating to parasitic diseases of sheep; but, from the wording of another paragraph, it is to be inferred that to a large extent reliance would have to be placed on the declarations of the owners that they had had the dipping carried out according to the prescribed regulations. Whatever may have been the intention of the Committee with regard to this point, there can be no doubt that, if an attempt were made to enforce simultaneous dipping, in most cases it would be impossible to obtain any evidence of compliance with the law except the declaration of the owner. The alternative of requiring the testimony of appointed inspectors regarding the dipping of thirty million sheep must be dismissed as altogether impracticable. In perusing the minutes of evidence we find that this difficulty was foreseen by some members of the Committee, and occasionally an attempt was made to lead a witness to express approval of compulsory general dipping by suggesting that a sheep-owner would never be so foolish as to evade a regulation manifestly enforced in his own interest. As an appreciation of the honesty and intelligence of the majority of farmers this may be quite correct, but at the same time it appears to overlook the existence of the anti-vaccinationist, and the possibility that an attempt to enforce sheep-dipping might bring to light the existence of a considerable number of " anti-dippers." Moreover, a little consideration will show that, if the farmer is as intelligent as he is generally supposed to be, he will in most cases be denied the incentive of feeling that the compulsory dipping of his sheep is in his own interest. Some of the witnesses pointed this out in quite a disconcerting way by asking why an owner who is perfectly certain that his sheep are free from scab should be compelled to carry out a procedure expressly designed to cure that disease. This is a question which probably 999 farmers out of every thousand in England and Scotland would be almost certain to put to themselves, and from that one may judge whether an annual dipping would be likely to be properly carried out without supervision.

But the whole of the difficulties in the way of securing simultaneous dipping of all the sheep in the country have not yet been considered. We have suggested that the " dipping period" might be fixed for August and September, and it is doubtful whether the period could be made much shorter without entailing great inconvenience in some

parts of the country. Now, it is absolutely essential that during this period all contact between dipped and undipped sheep should be prevented, and to that end it would be necessary throughout the whole country to prohibit the movement of sheep except when accompanied by satisfactory evidence that they had already been dipped within the prescribed period. Unless the traffic in sheep were to be paralysed during the dipping period, it is obvious that this requirement would throw on sheep owners and local authorities an amount of trouble and annoyance in excess of anything that has ever been created by the regulations dealing with other diseases. Here again we assume that something more than the mere declaration of the owner would be required as evidence that the sheep to be moved had already been dipped in strict compliance with the law.

Other difficulties than those we have here touched upon will occur to anyone who tries to think out the details of a scheme of compulsory general dipping, but we believe we have said enough on this head to show that it would be impossible to secure that all the sheep in this country should within a short period be "effectually" dipped.

In using the word "effectually" in the preceding sentence we had in mind merely the carrying out of the dipping operation in the manner prescribed by the regulations, that is to say, using a bath of approved composition and completely immersing every sheep for a minimum specified period. We have now, however, to discuss the question whether such a compulsory general dipping would, if it were practicable, be effectual in the sense that it would exterminate sheep-scab. It is important to notice here that, in the opinion of the Committee, a single dipping, when carefully carried out, is sufficient to cure scab. This is an opinion opposed to the experience of the four continents of the world, and therefore one naturally desires to know on what grounds it was adopted by the Committee. With regard to that point the report is quite explicit. If we may judge from the questions put to witnesses, members of the Committee at the outset of these enquiries shared the general opinion that two dippings with an interval of about a fortnight were necessary in order to bring an outbreak of scab to an end, but they were led to abandon this view by the results of certain experiments which were carried out under their direction. On turning to the account of these experiments which is embodied in the report, we find with surprise that the experiments designed to test the efficacy of a single dipping to cure scab were carried out on only eight sheep. The bare statement of this fact is sufficient to lead one to doubt the soundness of the conclusion which the Committee drew from the experiments, and the feeling of distrust is intensified by the complete absence of essential details in the meagre account which is given of the experiments in question. We are informed that the eight sheep were dipped with four different dips (two to each), and that three of these dips were

proprietary articles employed according to the directions of the makers. We are not told what was the age or extent of the scab lesions in any of these sheep, but from the evidence given by one of the witnesses (Q. 5538) it may be inferred that they were what the witness called " average cases of scab." One of these four dips contained as its active ingredients arsenic and sulphur, another tar acids and other tar products, another tar acids with paraffin, and the fourth tobacco and sulphur. It will thus be seen that the four dips found to be capable of curing scab with one dipping were representative of the dips generally used in this country, and hitherto universally believed to require two dippings in order to be efficacious.

We say that the result of the experiments with these four dips, employed on eight sheep, is the sole justification for the Committee's belief that scab can be cured by one dipping, but it ought to be stated that, in the opinion of the Committee, corroborative evidence was furnished by some of the other experiments in which sheep were dipped twice. A measure of the importance to be attached in this connection to these other experiments may be gathered from the fact that when no living acari were detected with the microscope in the interval between the two dippings it was thought permissible to conclude that the disease had been cured by the first dipping !

Although in the absence of important details in the account which is given of these experiments it is impossible to feel certain that the eight sheep were actually cured of scab by a single dipping, we may admit that they were without being compelled to abandon the view hitherto generally held that for the successful treatment of many outbreaks of scab two dippings are necessary. These experiments of the Committee, insignificant in point of number, were not required to prove that in many cases of scab one dipping is effectual. There is, we believe, plenty of authentic evidence to prove that thousands of sheep affected with scab have been cured by one dipping, but there is equally reliable evidence showing that one dipping, and even two or three dippings with the same materials, have failed to cure other cases in which the lesions were extensive and of old standing. It must therefore be held that the belief of the Committee in the efficacy of a single dipping to cure outbreaks of sheep-scab was rashly arrived at, and that any attempt to exterminate the disease by trusting to this method of treatment is fore-doomed to failure.

It is greatly to be regretted that this Committee surrendered the powers conferred upon them without carrying out the experiments that are undoubtedly necessary in order to make our knowledge regarding the action of sheep-scab remedies more precise. As already indicated, we believe that many cases of the disease are curable by a single dipping with almost any of the proprietary dips in general use, and also that some cases cannot be so cured even with the strongest anti-acaric agents that can safely be employed. Hitherto it has generally

been held that in dealing with outbreaks of sheep-scab the necessity for double dipping arises from the fact that the eggs of the acari are not with certainty destroyed by any dip that can be used without risk to the general health of the sheep. Under this view the rôle of the second dipping is to destroy the acari hatched out from the eggs during the interval between the two dippings, and there has been much discussion as to what this interval ought to be. So far as we know, however, this belief is based entirely on theoretical considerations, and it is quite possible that the unreliability of a single dipping is ascribable to the fact that, even when carefully performed, a single immersion sometimes fails to ensure contact between the whole of the acari or eggs and the parasiticide. This latter explanation will not appear far-fetched to anyone who takes into consideration the obstacles to the penetration of a watery liquid which are often offered by the length and thickness of the fleece, the abundance of oily matter at the surface of the sheep's skin, and the extensive crusts and scabs under or in which the parasites may be harboured in advanced cases of the disease. Lastly, it must be admitted that a single dipping may sometimes be efficacious in virtue of the amount of the active ingredients left in the fleece, and in dry weather persisting there to the destruction of newly hatched acari during the next few days; whereas in other cases the same treatment may fail because during a wet period the rain has freed the fleece of the residue of the dip. These are points which still remain obscure, and, as already said, it is unfortunate that the Committee missed an opportunity to clear them up which is not likely to recur soon again.

The last point that remains for consideration is perhaps the most important. Must sheep-scab be one of the perpetual plagues in this country unless a system of annual general dipping such as is outlined in the report of the Departmental Committee can be enforced? The recommendations of the Committee amount to a tacit affirmative in reply to that question. Fortunately, however, there is no need to take such a despondent view of the situation. We have given reasons for believing that the recommendations of the Committee are unworkable, and that they would fail of their purpose even if they could be carried out, but we have no hesitation in saying that there are other means, infinitely less irksome to the owners of sheep, by which sheep-scab could in a few years be absolutely eradicated. The persistence of sheep-scab in this country is not in the smallest degree attributable to the fact that the Local Authorities have lacked the power to compel the owners of healthy flocks to dip them. The disease has not been exterminated simply because the Local Authorities in the districts worst affected with it have not made full use of the powers already conferred on them. These are the districts which comprise the large, more or less unfenced, mountain and hill farms, especially in Wales and the Highlands of Scotland. Nothing is

clearer than that sheep-scab reigns in permanence in these districts, and that they annually supply the infected animals which cause a recrudescence of the disease in the lowlands every autumn. During the spring and summer months, when there is practically no movement of sheep from the hill farms, the disease almost disappears in the lowlands of England and Scotland under the regulations enforced by the Local Authorities. Evidence tendered to the Committee, notably that by the two witnesses representing the East Riding of Yorkshire, proved conclusively that no additional powers are required to enable a Local Authority to stamp out sheep-scab from its own district ; but the same evidence showed that the process of eradication has to be again and again repeated, owing to fresh introductions of infection from the districts of other Local Authorities who take no special pains to get rid of the disease.

The remedy for this unsatisfactory state of affairs is obvious. The Board of Agriculture should be pressed to undertake directly the task of suppressing sheep-scab in Wales, the Scottish Highlands, and other districts which constitute the distributing centres of sheep-scab. Compared to the difficulties which the Board has had to face in its efforts to exterminate swine-fever and pleuro-pneumonia, the task of stamping out sheep-scab in these regions would be a very light one. All that would be necessary would be the appointment of a moderate number of energetic inspectors, acting directly under the Board and exercising the powers of examination of sheep conferred by the Act of 1903. In every instance in which unreported sheep-scab is thus detected the owner ought to be prosecuted, and magistrates ought to be pressed to exact the maximum penalties allowed by the law whenever wilful concealment is proved. The prohibition of movement allowed by the Sheep-scab Order of 1898, applied to all sheep reasonably suspected of having been exposed to the contagion, and maintained until the Board are satisfied that the disease has been stamped out, would soon compel the owner to have resort to the dipping operations necessary to effect a cure. The gradually increasing penalties which in New Zealand were so successfully employed to compel the owners of diseased flocks to cure them would probably not be required in this country, where prohibition of movement would in less than twelve months itself constitute a heavy penalty. However, should experience indicate the desirability of strengthening the law in this direction, we fail to see why the Government should shrink from the necessary steps. In discussing this point it will be noticed that the Departmental Committee, with an inconsistency that is truly ludicrous, have stigmatised the method which eradicated sheep-scab from New Zealand as "too drastic a measure to commend itself to flock-owners generally at the present time." And this from a Committee which has declared that any outbreak of sheep-scab can be cured by one dipping! One marvels why anyone should shrink

from imposing increasing penalties on an owner who neglects to cure his flock of scab if that can be effected in the simple way laid down by the Committee. But one marvels still more when one reflects that, while the Committee think it would be too drastic to compel the owner of scabbed sheep to cure them—that is to say, to dip them once according to the Committee's own recommendations, they are unable to see any injustice in requiring the owners of perfectly healthy flocks annually to go through the procedure necessary to cure scab. We venture to say that if the Board of Agriculture were to endeavour to enforce an annual simultaneous dipping of all the sheep in the country, they would soon find it to be "too drastic a measure to commend itself to flock-owners generally at the present time."

PROTECTIVE INOCULATION AGAINST DISTEMPER.

ALTHOUGH canine distemper falls far behind many other animal plagues in point of economic importance, there is probably no contagious disease of the domesticated animals in which the public at large take a greater interest, owing to the simple fact that, if the domestic cat is left out of account, the dog-owners of the community greatly outnumber the possessors of animals belonging to any of the other domesticated species. The interest which the public take in this disease is, of course, also intensified by the fact that its victims are in most cases valued far beyond their actual worth in money. As a rule the death of a horse or an ox injures the owner in his pocket only, but the death of a dog often touches the affections of a whole household, and is felt as a loss for which the offer of another and more valuable animal of the same species would be no compensation. At the same time, it is not to be denied that the actual money losses annually inflicted on the country by canine distemper are of themselves sufficiently formidable to make it well worth while to search after some effective means of prevention. It must, we are afraid, be confessed that, in spite of all the researches and experiments that have been made in this direction, distemper is a disease which we are powerless to prevent, except by excluding the possibility of contagion. At any rate, the experiments recently carried out in London, mainly at the instigation of Mr William Hunting, would appear to have proved the worthlessness of the latest alleged specific against this disease.

It is no doubt unfortunate that some of these experiments failed to furnish data of a conclusive character, but even those that more or less miscarried have a certain value in indicating the very serious difficulties which must always be encountered in attempting to determine experimentally whether any method of vaccinating against dis-

temper is of real value. The chief difficulty arises from the fact that it is scarcely possible to gather together for experimental purposes any considerable number of young dogs and keep them free from distemper. This, of course, is owing to the almost constant occurrence of the disease in all except remote country places. It will be seen from the account of the experiments, which appears at a later part of this number, that after much trouble had been expended in obtaining the first series of puppies, thirty-three in number, the experiment had to be temporarily abandoned owing to the discovery that distemper had broken out among them. The same unfortunate accident occurred in connection with the second experiment, but the results of that experiment are not on that account altogether valueless.

It must be remembered that M. Phisalix has not only vaunted his vaccin as an almost certain preventive when used on healthy dogs, but has actually claimed that it exerts a beneficent influence when injected after the disease has declared itself, and has justified the systematic use of his vaccin in the case of dogs already attacked. Now, if there is any truth in the assertion that the vaccination of dogs already suffering from distemper exercises a favourable influence on the course of the disease, it is incredible that it should not have even a more favourable effect when it is used during the incubative stage. The results of this second experiment, however, lend no support to this view of the effect of the vaccination, for, as a matter of fact, a larger proportion of deaths occurred among the vaccinated than among the unvaccinated. In saying this we have assumed that all the vaccinated puppies were actually infected with distemper prior to the operation, though, from the date at which the symptoms set in in some of the fatal cases, it appears equally possible that they caught the infection at some date after the vaccination.

Another point of interest in connection with this experiment is the small number of fatal or severe cases among the unvaccinated animals. Putting aside the two which succumbed on the 15th July from some doubtful cause, only two deaths occurred among the control animals—a result which, we fear, some advocates of this form of vaccination would have regarded as a triumph for the vaccin if it had happened among vaccinated dogs. As in the case of many other diseases, the virulence of the distemper contagium, and the resistance offered to it by individual dogs, are very variable quantities, and on that account statistics which profess to give the measure of the value of vaccination by stating the proportion of vaccinated dogs which escape the disease, or suffer from it only in a mild form, are not deserving of serious attention.

The third experiment carried out by the Committee furnished results which cannot be explained away, and they will doubtless prove very disappointing to those who have for the past few years

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been carrying on a diligent propaganda in favour of the Phisalix vaccin. It is true that this experiment is open to the reproach that it embraced only a small number of puppies, but it was precisely to guard against the accident which vitiated the previous two experiments that the Committee resolved to be content in this case with a smaller number of animals. The experiment was carried out on two collie pups and two Irish terrier pups which were vaccinated with the Phisalix vaccin, an interval of fifteen days in the case of the collies, and of seventeen days in the case of the Irish terriers, being allowed to elapse between the two vaccinations. Subsequently the four vaccinated pups, in company with four controls belonging to the same litters as the vaccinated animals, were exposed to distemper infection. The first exposure took place twenty-two days after the second vaccination of the Irish terriers, and thirty-seven days after the second operation on the collies. A second exposure took place five days later. The puppies remained apparently well until the twentieth day after the first exposure, on which date one of them was found to be dull and not feeding well, and to have a distemper papule on the inside of one of its thighs. Curiously enough, this first animal to sicken was one of the vaccinated collies, and the next to show signs of illness was the other vaccinated collie. The two control collies subsequently developed the disease, and one of them eventually recovered, while the other was killed when its condition appeared hopeless.

The result of the experiment so far as the collies were concerned was thus altogether to the discredit of the vaccin. The two vaccinated animals were the first to be attacked, and both died from uncomplicated distemper, while one of the unvaccinated controls recovered. It would, of course, be unjustifiable to suggest that the operation had actually favoured the fatal ending in the case of the two vaccinated collies, but the result of the experiment makes it impossible for anyone to doubt that the vaccination had failed to confer any useful degree of immunity against distemper. Most people will probably be inclined to put the failure in stronger terms, and hold that the vaccination conferred no protection at all.

In the case of the Irish terriers the result of the experiment was also entirely unfavourable to the vaccination. One vaccinated and one unvaccinated pup contracted and died from uncomplicated distemper, but the other two animals—one vaccinated and one control— survived, and during the course of the experiment it was never possible to affirm that these two pups had contracted the disease at all. This latter fact is very significant, for, in view of the fate that befell the other two pups belonging to the same litter, it is impossible to believe that any of these animals had previously suffered from distemper. Nevertheless, these two animals appeared to escape the disease, although for two months they were never out of a loose-

box in which during that period six other animals died from dis-temper. The apparent immunity of these two pups furnishes a fresh illustration of the fallacy to which one is exposed when vaccinated dogs escape the disease and the result is placed to the credit of the vaccination. This chance of error may doubtless be eliminated, but to that end the experiment must be so planned as to exclude the possi-bility (1) of a previous attack, and (2) that the animal may have possessed a high degree of natural immunity. As a matter of fact, the latter possibility cannot be altogether excluded by any precau-tion, but it may be reduced to the vanishing point by having a sufficient number of control animals belonging to the same litter as the pups selected for vaccination.

We do not believe that the vaccin prepared by M. Phisalix confers any protection whatever against distemper. Furthermore, we do not believe that the organism which M. Phisalix regards as the cause of distemper is anything more than an occasional accidental accompani-ment of that disease. In our judgment the etiology of canine dis-temper, in the narrowest sense of the expression, is still an unsolved problem.

Reviews.

Handbook of Meat Inspection. By Robert Ostertag, M.D., Professor in the Veterinary High School at Berlin. Translated by E. Vernon Wilcox, M.A., Ph.D., London : Baillière, Tindall, and Cox, 1904.

PROFESSOR Ostertag's text-book on Meat Inspection is universally accorded the first place among works on that subject, and the English translation of it which Dr Wilcox has been at the pains to provide is sure to prove welcome to all those who in English-speaking countries are engaged in the inspection of animals slaughtered for food purposes. As a guide to meat-inspectors outside the limits of the German Empire the work is open to the objection that it is written from the strictly German standpoint, and is therefore encumbered with a certain amount of matter which is not of direct interest in other countries. It may be surmised that, even in Germany, at least veterinary inspectors of meat find a not inconsiderable part of the work superfluous. Practical meat-inspection is almost entirely an application of the known facts of veterinary pathology with the object of excluding unsound and unwholesome animal food from the -market, and at the present day the veterinary student, unless his training in pathology has been sadly neglected, approaches the study of meat-inspection with a great part of the necessary knowledge already in his posses-sion. But, unfortunately, in many countries the inspection of meat is still partly in the hands of persons who have not had any veterinary training, and therefore whoever essays to write a work on the subject must not assume that his readers possess any knowledge of the anatomy or pathology of the domesticated animals.

When this is remembered, no surprise need be felt that the work now under review is a rather formidable volume, extending to nearly 900 pages. It is, in fact, a text-book of veterinary pathology and bacteriology, in which almost every morbid condition to be met with in the animals slaughtered for food purposes is considered from a special point of view, viz., its effect on the part or organ concerned as an article of human food. Over and above this, the work deals with the laws governing meat-inspection (in Germany), the "art of butchering," the appearance of the healthy organs and tissues, the anatomical features serviceable for the recognition of the flesh and organs of different species of animals, the adulteration of such materials as sausages. the preservation of meat, and the methods of sterilising or otherwise disposing of condemned meat. In short, it may safely be said that, while some meat-inspectors may find in it more than they require, no one is likely to have to complain that anything bearing on the work of meat-inspection has been omitted from consideration.

What has just been said refers to the handiwork of the original author, but the translator's share of the work also calls for notice, and we have regretfully to express the opinion that here there is room for some improvement. The indispensable qualifications of a good translator are (1) a competent knowledge of both languages, and (2) an intimate acquaintance with the subject matter of the work to be translated. We have come across some errors which appear to indicate that Dr Wilcox does not possess the second of these qualifications in a very high degree. The following are two examples. In the paragraph dealing with the lesions of glanders (p. 597), it is said: "In 52 cases examined by Bollinger, the lungs were affected in only 4 cases, and in only 10 of the 216 *post-mortem* examinations made in the Berlin High School." Needless to say, this is an error not found in the original work. The sentence would be correctly rendered if the words "free from disease" were substituted for "affected." Again, it is said to be "a remarkable fact that in generalised tuberculosis of young cattle (up to four years of age), the spleen is almost always affected and the kidneys are free from tuberculous foci, while in older cattle the kidneys, together with the spleen, uniformly show tuberculous alterations" (p. 623). Here the words "together with the spleen" should read "and not the spleen." Tuberculosis of the spleen of the adult ox is an extremely rare lesion, and a very little practice in meat-inspection would have saved the translator from the absurdity of representing it as a constant lesion.

Friedberger and Fröhner's Veterinary Pathology. Translated and edited by M. H. Hayes, F.R.C.V.S. Vol. I. Fourth edition (revised and enlarged). London : Hurst and Blackett, Ltd., 1904.

A MELANCHOLY interest attaches to the new edition of this work owing to the death of the translator since the date of its publication. In speaking of the previous edition, we have strongly recommended the work as a thoroughly scientific treatise on the diseases of the domesticated animals, and on this occasion it need only be said that the new edition is superior to its predecessor, as the translator has endeavoured to bring the matter up to date, by adding a chapter dealing with surra, tsetse-fly disease, South African horse-sickness, and a few other diseases which were not dealt with in the previous edition. It may be remarked that the title of the work is a little misleading, as it is in reality a treatise on the practice of veterinary medicine, although in most cases the pathological anatomy is more fully described than is customary in books mainly designed for the practitioner. The present volume represents only about half of the original German work, but it is to be hoped that a translation of the remainder will soon be made available for English readers.

Lehrbuch der allgemeinen Pathologie für Tierärzte und Studierende. Von Professor Dr Th. Kitt, in München. Stuttgart: Ferdinand Enke, 1904.

PROFESSOR Kitt, in common with all other teachers of veterinary pathology, has felt the want of a text-book dealing with the general pathology of animal diseases, and he has thereby been stimulated to produce the present work. It extends to 432 pages, and contains 119 figures in the text, as well as a number of excellent coloured plates. After a short introductory chapter, the author discusses at considerable length the subjects of predisposition and immunity, following which are chapters devoted to congenital and inherited diseases, the causes of disease, the course and termination of disease, circulatory disturbances, degenerations, reparatory processes, tumour formation, and functional disturbances. Everywhere the author displays a wide knowledge of veterinary pathology and the power to expose it for the benefit of the student. The present work may be said to form the complement to the author's previous text-book on the pathological anatomy of the domesticated animals, and it constitutes a useful addition to veterinary literature.

Mammalian Anatomy, with special reference to the cat. By Alvin Davison M.D., Professor of Biology in Lafayette College. London: Rebman, Ltd., 1903.

THIS is a handy volume of 242 pages, and it has been designed to serve as a guide to students of zoology where the cat is selected for dissection as a vertebrate type. It appears to be very well adapted for this purpose, as the structure of the cat's body is described with just that degree of fulness which is likely to prove most useful to the student of zoology. The illustrations, 108 in number, are uniformly excellent.

Tuson's Veterinary Pharmacopœia, Sixth Edition. Revised and edited by James Bayne, F.I.C., F.C.S. London: J. & A. Churchill, 1904.

IN preparing a new edition of this little book, Mr Bayne has effected some improvements on the previous one by eliminating a number of errors and omissions. Some alterations have also been made with the object of bringing the work into line with the 1898 edition of the *British Pharmacopœia*. Although the work is said to be designed for both, it must be confessed that it appears to be much more likely to meet the requirements of the student than those of the practitioner At the same time, even those who are no longer students will find it handy for reference in the pharmacy.

CLINICAL ARTICLE.

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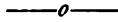
DEATHS BY ELECTRIC SHOCK.

By R. LEWIS GREEN, M.R.C.V.S., Dudley.

HAVING had the opportunity of making *post-mortem* examinations of three horses killed in this way, and thinking it would be of interest to the profession as showing the extensive and deep-seated injuries that can be caused by burns by electric fluid, I have been induced to record the history of the cases.

A pole placed in the middle of the road which carries the overhead wires for tram-car traction one wet day last winter became " alive." As a waggon loaded with bar iron, drawn by three horses, viz., one in the shafts and two in traces leading, was nearing this pole, the leaders, according to the driver, appeared to be involuntarily drawn towards it. The leading horse came in contact, and dropped dead instantaneously. The next trace horse behind fell dead without a struggle, the current evidently passing along the chain traces and on again to the shaft horse, which also fell and died in a few seconds. The ground, being moist, was charged with " fluid " for a distance round. The driver, who was sitting on the waggon, received a shock in the neck and arms, the current passing up the wet reins.

White Mare. Eight Years Old.

This animal was in the shafts. On the off thigh were two lineal burns as if produced by a wire or chain, each 10 by 1½ inches in extent. Burnt area on the off hind leg, 5 inches in circumference; another area on the off side of the abdomen, 9 by 1¼ inches wide, and another burn on the off arm, 2 by 1¼ inches.

On the off shoulder of this animal was a burn which extended through the skin into the muscular tissue beneath, this one being 1 inch deep, and 1¾ by 1½ inches in surface extent.

Several areas on the off side of the neck showed burns of more or less severity. The trace chain was probably the cause of a burn which extended from the right nostril over the lips, to the under portion of the jaw, and was 15 by ½ an inch in size. Behind the near shoulder were two areas, the one 7½ by 2½ inches, the other 5½ by 2 inches. These were probably due to the metal work on the shaft and the back chain. On the left shoulder was a hole burnt through the skin, 1 by ½ an inch in extent, which corresponded to the length of a trace chain link. The near side of the neck had a scorched area 5 by 2 inches. The hair of the chest was singed, and the surrounding tissues were more or less affected. The tongue and gums were scorched and of a purple colour, and undoubtedly the bit was the conductor which caused injury to these parts. The pectoral muscles of

this animal were of a purplish tint, and exuding from their substance were small hæmorrhages about the size of a pea, which were semi-fluid and of the colour and consistency of tar. The adominal fat corresponding to the burnt area of the skin of that region was of a purplish colour.

Spleen.—This organ was firmer in texture than normally, very purple in colour, with areas of raised petechiæ on its outer surface. On making an incision into its substance, it was found to be engorged with the characteristic semifluid tarry blood.

Liver.—Several small " milk spots " were to be seen on the surface of each lobe. It was very firm to the touch, and, on incision, purplish blood oozed from it, but at the base of each lobe, extending probably 4 or 5 inches over its outer surface, and confined to the " capsule, were several black or purplish-black areas, varying from the size of a pin's head to 1½ inches square. This lesion in the liver I have never noticed before.

Heart.—This organ, as in the majority of cases of death from " electrocution," was flabby to a very great extent, but an *ante-mortem* clot was present in the left auricle and ventricle. It is therefore quite evident this animal did not die instantaneously, but, from the fact of her being in the shafts, she did not come into contact with the pole at all, her death obviously being caused by the current traversing the trace chains.

Stomach and Intestines.—The blood vessels of these were distended and filled with blood of a semifluid, tarry nature. Each one could be traced its entire course along the organs. Several portions of the small intestine, varying in length from 6 inches to 10 inches, were considerably contracted. In all probability these contractions corresponded with the burnt areas of the abdominal region.

Black Mare. Nine Years Old.

This animal was in the middle of the team. The left side, along which the trace chain rested, was burnt badly, and there were seven holes, corresponding to seven links of chain, on the shoulder. The off side also was extensively burnt and lacerated. An area on the hip, 6½ by 2¾ inches. A patch on the ribs of this side, 9½ by 9 inches, in the middle of which was a deep wound 2 inches long, which penetrated the intercostal muscles, scorching the pleura and lung, as described below. Also twelve link marks, two down the arm, and ten very deep ones between the stifle, which joint was opened, and the posterior portion of the thigh. The off thigh had three lineal burns, as if a hot rope had been drawn across it ; these were 8½ by 1½ inches, 5 by 1¼ inches, and 13 by 1¾ inches, respectively. A burnt area in the middle region of the neck extended from one side round the trachea and upwards ; it was about 30 inches in length, and undoubtedly a portion of this was caused by the chain on the end of the leading rein.

Spleen.—Firm, of a deep purple tint, with raised petechiæ on its surface ; on incision semifluid, dark-coloured blood oozed slowly from it.

Lungs.—*Right :* the apex and a large area of the middle of this lung were emphysematous. *Left :* a patch 3 inches square near the

base and in the thick portion showed a decidedly burnt surface, the lungs were covered with petechial spots, and both were gorged with the before described blood.

Brown Gelding. Eight Years Old.

This animal was leading, and, although it received the shock direct from the post, it was not burnt so extensively as one would have expected.

There were scorched areas as follows on the near side, where the skin resembled burnt leather, as under : one on the shoulder 4 inches in circumference, middle of ribs, 7 by 6 inches, flank, 8½ by 2½ inches, hip, 1 inch square, a large triangular area on thigh, the sides being 10, 8, and 11 inches long respectively. Tibial region, a burn 18 by 1 inches, running right round to the inner side of hock ; this was caused, in my opinion, by the horse falling as a cow lies down, with his hocks and buttocks resting on the ornamental base of the post. The abdomen on this side had a patch 6 inches square.

Stomach and Intestines.—The blood vessels were mapped out to a remarkable degree as in the other two animals ; a portion of small intestine, to the length of about 11 inches, was contracted, probably corresponding to the burnt area of the abdomen.

Liver.—This organ was firm with blood and in the same condition as previously described.

Lungs.—Marked petechiæ on both lobes, blood exactly as in the other animals.

Heart.—Flabby, collapsed, and devoid of blood.

Kidneys.—Capsules easily removed in each animal.

Spleen.—Normal in consistency.

Remarks.—The majority of skin burns were distinctly visible on the under surface when the skin was removed, and in many cases the areas were clearly defined on the muscular tissue immediately beneath. The hide when burnt cannot be neatly skinned, and in every instance the muscular tissue corresponding to a burnt surface above is charged with this dark semifluid blood. The cutaneous vessels and jugulars in each subject were gorged with blood. The pupils of each animal were dilated fully. The necks of two animals had a marked curvature, and could not be straightened. One animal died with the tongue between the teeth, the lower jaw being drawn to the left. In the gelding the penis was pendulous. The anus of each animal was dilated, the rectum was forced towards the orifice, but did not protrude, and the mucous membrane was of a purplish tint. The vaginæ of the mares were not thus affected. There was a frothy, highly crimson-coloured, bloody discharge from the nostrils of each animal. Severe rigors were noticed in each animal on receiving the shock.

It is remarkable how small a voltage will cause the death of a horse, viz., 100 volts, whereas the human subject may withstand 500 or more volts.

𝔄bstracts and 𝔎eports.

PROTECTIVE INOCULATION AGAINST ANTHRAX.

[HERR. O. HEINE having published certain conclusions regaiding the combined method of conferring immunity against anthrax recommended by Professor Sobernheim, the latter replies in defence of his own views. The discussion bears on certain experiments carried out on a large estate in a place designated by the letter E——].

Professor Sobernheim considers that the results at E——, where a large number of animals showed more or less severe reactions and where some died, are of a quite exceptional character.

Although when this method of protection was first practically used a few cases reacted severely and some were lost, no great difficulty was experienced in preventing a repetition of such occurrences. They were due to circumstances which are now better understood, and which observation in the laboratory and in practice have enabled the Professor to overcome. At the commencement he personally supervised the preparation of the serum and cultures required by his method, and either personally carried out or supervised all the inoculations, but owing to the extension of the method it soon became impossible to provide such close personal supervision, a fact which will enable any competent and unbiassed investigator to understand why certain new difficulties arose. The precautions required in the preparation, despatch, and storage of the cultures were of great importance and demanded special attention, but in addition the numerous important observations then made showed that cattle exhibited much greater variations in relation to the new method than was formerly believed, depending on their species, race, feeding, work, etc. In this connection it was found that working oxen showed very much less resistance than others, and also that severe work rendered them still more sensitive. As illustrating this, forty working oxen which were inoculated at the same time as a large number of other animals showed severe reactions and three died, whilst, of an equally large number of cows inoculated on the same day, with similar material, by the same veterinary surgeon, all remained completely healthy.

The earlier losses happened at a time when the above-mentioned facts were imperfectly understood, and were incurred on three farms where about forty, sixty, and one hundred and thirty animals, respectively, were inoculated. On the first, three, on the second, four, and on the last farm, two animals died, a total of nine, almost all of which were working oxen. As a result of this experiment and in the belief that such accidents could best be avoided by using less virulent cultures and not by increasing the dose of serum, the virulence and dose of the culture material were afterwards slightly diminished, and precautions were taken in preparing the cultures to render them as regular in efficiency as possible. The immunising power of the method as shown by experiments and actual practice was not appreciably influenced by this trifling diminution in virulence of the culture.

The result has been that for eighteen months past all the inoculations have proved successful. As reported elsewhere (*Deutsche Med. Wochenschr*, 1904, Nos. 26 and 27), more than 60,000 cattle, sheep, and horses were inoculated without any dangerous reactions and without any loss. Since then a further very large number of inoculations have been carried out (more than 40,000

between the middle of March and the middle of June) without a single really bad result. Most of these were practised in South America (the Argentine and Uruguay), though several thousand animals were inoculated in Germany.

It need scarcely be said that in dealing with large numbers an animal here and there may react somewhat severely, and it is perhaps not without practical interest to mention that some animals have shown urticaria-like symptoms soon after inoculation. Probably this was due to the injection of "foreign" serum (the serum employed was usually a mixture of horse, sheep, and ox serum). A similar reaction has often been seen and described in man after the use of diphtheria serum.

A method which has been tested under the most varying circumstances and in the most widely separated regions on more than 100,000 animals must be allowed to have proved its safe character. The unfortunate results of the inoculations in E—— during March of this year cannot therefore be regarded as characteristic of the method, but as a quite exceptional occurrence and a misfortune. As a result of the enquiries and careful tests which were at once instituted it seems unquestionable that the culture used for inoculation was of too high a degree of virulence, and was in fact the cause of all the resulting symptoms. How such a culture came to be employed cannot, unfortunately, be clearly proved, but it is of interest to know to what extent a recurrence of the misfortune is preventible. Professor Sobernheim believes that such an accident need never again occur, and declares that cultures can always be obtained of the exact required degree of attenuation, grounding his opinion on the large number of inoculations which have already taken place.

Heine, however, has stated that the experiments carried out on the initiative of Professer Ostertag in E—— showed the serum to be inefficient, and he is inclined to ascribe the unfavourable course of the experiments to the use of too weak a serum. This, however, Professer Sobernheim emphatically denies. He considers this view is, a priori, discredited, because a culture of normal attenuation would scarcely produce such a severe reaction even if injected alone, without any serum. The course of affairs in E—— can only be explained by the cultures possessing too high a degree of virulence, so high as to be uninfluenced even by the most active serum, in the above-mentioned doses. That the serum was not too weak, however, is shown by the fact that exactly the same serum was used before and afterwards on about 4000 animals, with excellent results. Moreover, a careful test was undertaken by Professer Sobernheim in the presence of a representative of the State Institute for Experimental Therapeutics (Frankfurt on Main) and proved the serum to possess a markedly specific action.

Of five rabbits previously inoculated with the serum, three resisted infection with viruleut anthrax cultures, whilst two others long survived the control animals. This testifies to a degree of efficiency which must be regarded not only as considerable but as fully sufficient. Professor Sobernheim believes that the result completely justifies the claims which he has advanced as regards the protective value of anthrax serum for practical purposes.

Heine's general conclusions deserve some attention. He believes that the combined method of inoculation against anthrax as recommended by Professor Sobernheim can give protection, but that anthrax serum is incapable of curing infected animals even when large doses are subcutaneously injected at the commencement of the disease. Sobernheim fully accepts the first conclusion but differs from the second. He regards it as natural that here and there an inoculated animal may subsequently die from anthrax, and claims no absolute immunity for inoculated animals. Such occurrences, however, he regards as exceptional, and as of little account when compared with the great number of favourable results obtained.

Regarding Heine's refusal to accept anthrax serum as a curative agent he

has more to say. In order to prevent misunderstanding he draws attention to the fact that of the eight animals that died five were not treated with serum at all, viz., three oxen, one heifer, and one small bull. Of the three other animals two oxen had received 80 cc. of serum, whilst it was impossible to learn with certainty whether the last animal, a small bull, had or had not received any serum. But even including this animal, only three of the eight victims had received an injection of serum, whilst, on the other hand, thirty to forty other animals, which showed more or less severe symptoms of illness and were at once treated with serum, survived.

Among those which suffered most severely were the eight working oxen, whose temperature Heine gave in tabular form.

Sobernheim draws attention to the fact that the temperature of these animals was only taken once a day instead of every two hours, and even then was not invariably noted at the same period of the day. These temperatures therefore only indicate the degree of fever twenty-four hours after an injection of serum.

Sobernheim is quite unable to understand Heine's commentary on the temperature records, viz., "that in no case could any marked diminution in temperature be detected as a consequence of the injection of the anthrax serum even when large doses were employed." A glance at the table shows how little justified was this conclusion. In two cases the temperature at once began to fall after the serum injection, and in three to four days reached normal. In two other cases the temperature fell to a considerable extent immediately after the first injection, and, although a slight rise again occurred on the third day, the temperature soon fell to normal. In a further case the temperature steadily remained within normal limits before and after the serum treatment although the animal was severely affected and showed œdematous infiltration. Only in the three cases did the serum apparently fail to affect the course of the fever, and Sobernheim, whilst again emphasising the incomplete nature of the temperature records, declares his belief that in by far the greater number of cases they show that serum injection checked the course of the fever, and in some cases reduced it from a very high point to normal within a few days.

It is at least undeniable that the eight animals which are supposed to have proved the unreliability of the serum treatment survived, and that was really the sole object of the entire treatment. Sobernheim is firmly convinced, and claims to be supported by the experts who witnessed the experiments and even by Heine, that without the use of the serum, or more exactly, without treatment, several of the severely diseased working oxen and other diseased animals would certainly have died.

Leaving personal impressions out of account, however, the fact remains that thirty to forty animals suffering from anthrax were treated with serum, and that although three died the others survived.

Regarding the method of using the serum, Sobernheim mentions that he only learned of the accidents at a very late hour, and that when he arrived in E—— six animals had already died, two of which had received 80 cc. of serum each. He therefore thought it advisable to employ very energetic treatment, and with the exception of the before-mentioned small bull succeeded in preventing any more deaths. He was obliged to increase the dose to 100-150 cc. having found that injections of 80 cc. had in two cases failed to cure the animals. Many of the patients had certainly not been treated "at the commencement of the disease," but only a day or two after the appearance of symptoms.

Sobernheim's previous experiments convinced him of the need of repeating the injection, as he had found that single injections were not always successful, nor was the apparent improvement always lasting.

He thinks it might have been possible to succeed and to cut short the

process by injecting smaller quantities intravenously, but he abstained from this method because he had had no personal experience of it and was loth to employ it for the first time in such critical cases. (*Berliner Tierärztl. Wochenschr.*, No. 34, 18th August 1904, p. 577.)

PROTECTIVE INOCULATION AGAINST SWINE ERYSIPELAS IN WURTEMBERG IN 1903.

DURING 1903, 39,578 swine were protected by the Lorenz' method of inoculation. In previous years the numbers were as follows: 1902, 27,811; 1901, 20,801; 1900, 15,217; 1899, 12,501; 1898, 9093; 1897, 7178, 1896, 1487; 1895, 63; 1894, 136; 1893, 9. Of these, 37,275 animals, distributed in 60 districts and 585 sub-districts, were inoculated by government officials, whilst 2303 were inoculated by private practitioners.

Forty-four of these animals were further subjected to curative inoculation. Thirty-one recovered, five died, and two, whose recovery was doubtful, were slaughtered. The remaining six were slaughtered without any pressing reason. In those which died the disease was already far advanced when curative inoculation was practised.

Of the 39,534 protected swine 1781 had been inoculated with serum during the previous year, and only received culture injections during the official year now under review. In 373 cases culture No. 1 alone was given, and in 1408 cases culture No. 2 also. Of the other 37,753, which were simultaneously inoculated with serum and culture No. 1, 21,401 also received the second cultural injection. Although a number of swine (of which particulars are given) died during the course of the inoculation, no losses directly traceable to inoculation occurred. No case of conveyance of swine erysipelas from inoculated to uninoculated swine was recorded during the official year. The inoculations as a whole must therefore be regarded as exceedingly satisfactory.

The degree of protection left nothing to be desired, for up to the close of the official year no case of swine erysipelas in an inoculated pig had been proved. Three swine, which died after the lapse of the time necessary for inoculation reaction, were believed by the owner to have suffered from swine erysipelas; in one case this view was negatived by a bacteriological examination of the spleen. Another pig had suffered from an intercurrent disease but had recovered.

The serum and the necessary cultures were prepared in the Hygienic Laboratory of the Royal Würtemberg Medical College, which also undertook the sterilisation of the required syringes.

The inoculations carried out by state officers consumed 210,464 litres of serum, an average quantity for each pig of 5·9 cc. The quantities previously used were as follows: 1902, 5·7; 1901, 5·9; 1900, 6·1; 1899, 6; 1898, 5·9; 1897, 5·8; 15 701 litres of serum were supplied to private veterinary surgeons.

The entire outlay for the inoculations performed by officials, including the fees of the sixty-eight veterinary surgeons (each of whom received a living and travelling allowance, as well as 15 pf. per animal inoculated), amounted to 20,214 marks 54 pf. After deducting fees paid by the owner, amounting to 17,318 marks 5 pf., the cost to the state was 2896 marks 49 pf.

On an average therefore each inoculation cost 54·2 pf., of which the state paid 7·8 pf.

During the official year a system of insurance was instituted against losses due to inoculation. Compensation was made entirely contingent on the

owner delivering the necessary internal organs at latest three days after the animal's death for the purpose of bacteriological examination. Only twenty-four demands for compensation were made. Careful bacteriological examination of the organs forwarded, comprising microscopical examination, inoculation of mice and pigeons, and cultivation on artificial media, showed, however, that in twenty-three of the cases death had not resulted from inoculation.

In the twenty-fourth case no satisfactory distinction between swine erysipelas and mouse septicæmia bacilli could be made on account of the advanced state of decomposition of the organs sent. As the animal from which these organs had been obtained had died at least four days previously this demand was also dismissed. (Beisswänger, *Berliner Thierärztl. Wochenschrift*, No. 33, 11th August 1904, p. 565.)

THE TREATMENT OF TORSION OF THE GRAVID UTERUS IN COWS.

In the *Berliner Archiv* for 1902 Lempen gave a summary of the extensive literature dealing with this disease and of the varying views held regarding its origin and treatment, particularly as to the direction in which the animal should be rolled in order to reduce the torsion.

In common with the majority of authors, Lempen rightly concluded that the rolling should be in the same direction as the torsion. He also proposed to describe the torsion as being to right or left according to the direction of the spiral folds to be found on the upper wall of the dilated cervix uteri when the examiner stands behind the animal. This mode of describing the changes seems least likely to cause misunderstanding.

Haase divides his cases into three groups according to the method of treatment employed. The first comprises cases where delivery was effected without rolling or hobbling the animal, which was either standing or lying down.

The second, cases in which delivery was only possible after rolling, and

The third, two cases which Haase himself did not treat but only examined.

In describing the degree of torsion he takes as his index the upper wall of the uterus. Where this has moved through an angle of 90 degrees he speaks of quarter torsion; when through an angle of 180 degrees (in which case the upper wall will have become the lower) of half or semi-complete torsion; when through an angle of 270 degrees as three-quarter, and when through 360 degrees (the upper wall having then described an entire circle and returned to its former position) as complete torsion.

In forming a diagnosis, the extent to which the maternal passages seem fixed in position, the amount of resistance they offer to the hand, and the degree of tension in the spiral folds, to some extent indicate how far torsion 'has proceeded. Where the spiral folds are very tense and the passages completely immovable, so that the operator cannot reach the fœtus, torsion is usually complete; in cases of less complete torsion (one-quarter to three-quarters) the cervix uteri is closed and displaced to a proportionate extent, and the resistance to the passage of the hand is in keeping.

Case No. 1.—Primipara. A three-year-old Simmenthaler cow had shown pains for some hours without labour being completed. On manual examination moderately tense folds running towards the right could be felt on the upper wall of the dilated cervix uteri, so that it was necessary to bend the arm at the wrist and avoid the folds in order to pass the hand into the uterus. The calf still lay in the unbroken membranes, with the head and fore limbs directly behind the obstruction. There was therefore moderate torsion between one

quarter and one third of a revolution, towards the right. Haase broke the membranes, and, after allowing a portion of the fluid to escape, secured the fore-limbs and head with straps, and by careful traction delivered the calf alive. Slight bleeding from the vagina followed. On the lower wall of the uterus, at the point where it becomes continuous with the cervix, was a rupture nearly as large as a cheese plate, implicating, however, only the mucous membrane. This was due to the lower wall having been pressed against the anterior edge of the os pubis. Lysol injections were made daily, and the wound healed in about a fortnight. The cow and calf remained healthy.

Case 2 was that of a black and white Holland cow, which had travelled a considerable distance by rail and had afterwards been forwarded by cattle van. It had shown labour pains without, however, the calf appearing. On turning the animal round slight crepitation could be heard. The right hip seemed lower than the left, giving the pelvis an oblique appearance. Fracture of the pelvis was suspected without its exact position being diagnosed. Semi-complete torsion of the uterus was revealed on careful examination. No attempt was made to roll the cow because of the severe injury already existing. By cautious traction on the hind limbs the calf was delivered but proved to be dead. Moderate bleeding from the vagina followed. The animal died two days later.

Post-mortem examination proved it to have suffered from luxation of the left sacro-iliac joint, the two surfaces being separable, a fact which explained the crepitation and oblique position of the pelvis during life. The upper wall of the uterus also showed a torn wound about 8 inches long communicating with the peritoneal cavity. The peritoneal cavity itself contained blood-stained fluid and a large blood clot. The animal had died from septicæmic peritonitis. Haase considers that both the luxation of the sacro-iliac joint and torsion of the uterus were probably caused by a single violent shock, such as a sudden fall or severe concussion produced in shunting the cattle truck.

Case 3 was that of a young Simmenthaler cow with slight torsion towards the left, which Haase estimated at one quarter to one third. By cautious traction on the fore-limbs the young animal was safely delivered. Cow and calf remained healthy.

Case 4 was that of a primipara of Simmenthaler breed which showed one-quarter to one-third torsion towards the right. By slow cautious traction on the hind limbs the calf was brought into the maternal passages, but complete delivery necessitated the use of considerable force. The calf was dead. It weighed nearly 120 lbs., and was therefore rather above normal size. The mother escaped rupture but showed severe inflammation of the vagina and cervix, which subsided in about fourteen days, after frequent irrigation with 1 per cent. lysol solution.

As shown by the above cases, Haase was partially successful in delivering animals suffering from torsion of the uterus without casting them, particularly in Cases 1 and 3.

He had greater difficulty, however, in effecting delivery where rolling proved necessary in order to reduce torsion. Being uncertain as to the direction in which to roll, he in some cases tried first one and then the other. Only after considerable experience did he discover the correct direction.

Case 5. An adult cow of Simmenthaler breed and in very good condition had shown labour pains for ten to twelve hours without delivery being effected. Semi-complete or complete torsion of the uterus was diagnosed. The cervix was firmly constricted. The spiral folds were extremely tense, so that manual examination was only possible by the exertion of considerable strength, and by strongly flexing the wrist the fore feet and head of the calf could be felt.

The owner having been informed of the gravity of the case, the animal was hobbled and rolled on a smooth horizontal floor thickly covered with

straw. During a period of one and a half hours about twenty turns in either direction were given. The torsion was not reduced in the slightest degree.

As the owner strongly opposed slaughter and, against Haase's advice, demanded that attempts at delivery should be made despite the torsion, Haase placed cords on the front legs. He was unable to pass a cord round the neck, but inserted a hook into the orbit. By employing considerable force whilst the cow was standing the calf was extracted but proved to be dead. The mother appeared to have survived the operation well, but three or four minutes later shuddered severely, lay down, and died. Death had resulted from bleeding into the abdominal cavity.

Case 6. Primipara with half to three-quarter torsion of the uterus. The animal was turned about fifteen times in either direction, but, no diminution occurring in the torsion, it was slaughtered.

Case 7 was that of a cow which had borne several calves. Having shown labour pains for two days, the animal was examined and found to be suffering from half to three-quarter torsion of the uterus, towards the left. After casting it was rolled about twenty times in either direction, but without the torsion being reduced. Before slaughtering, Haase determined to try a new method. The animal was lying on its left side and was rolled *down-hill* on to its right side. The torsion seemed at once to have been reduced, and the calf was born alive, though it died about ten minutes later. The uterus showed an incomplete rupture as large as a cheese plate in the region of the anterior margin of the os pubis. The rupture did not communicate with the peritoneal cavity, but three days later the cow died of puerperal septicæmia.

From the results of this case Haase concluded that the rolling should be performed on a sloping bed and in the direction of torsion.

Case 8 showed semi-complete torsion towards the left. The animal was hobbled and placed on its left side. A sloping bed of straw was made, extending backwards from the level of the crib to the stable drain. On this the animal was laid on its left side and rolled over once down-hill, so that it came to rest on its right side. The torsion was immediately reduced and delivery effected. A penetrating rupture of the upper wall of the uterus, however, was detected, and the animal was slaughtered.

Case 9 was that of a cow with semi-complete torsion towards the right. A bed was prepared as above described, the animal laid on its right side, and rolled down hill on to its left side. The torsion in this case was also reduced ; the calf was delivered but only lived fifteen minutes. The cow suffered no injury and remained healthy.

Case 10 was that of a Simmenthaler cow in good condition. She had previously borne several calves and had no difficulty in delivery. As labour pains were not followed by the appearance of the calf, the neck of the uterus was examined and found to be thickened. No constriction could be detected, nor were spiral folds present.

As this condition persisted for several days, and the animal began to show fever, loss of appetite, and tympanites, the case was regarded as torsion of the uterus and the animal was slaughtered. *Post-mortem* examination showed torsion of the anterior portion of the gravid cornu of the uterus. Examination per rectum might possibly have enabled this condition to be detected during life.

Case 11 was that of a well nourished Simmenthaler cow which showed labour pains without delivery following. Semi-complete torsion of the uterus towards the right was diagnosed. Haase recommended attempting reduction, but the owner opposed this and called in another veterinary surgeon. The calf was delivered dead and the cow was at once slaughtered.

Considering in detail the circumstances attending this disease in cows, the

uterus assumes the appearance of a pendulous organ the body and horns of which constitute the bob of the pendulum, whilst the ligaments represent the cords by which it is suspended. The fixed points are formed by the insertions of the two ligaments in the neighbourhood of the two external angles of the ilium.

The uterus, however, is also steadied in position by the vagina and by the cellular tissue surrounding it; in fact, in non-pregnant animals it can scarcely be regarded as pendulous, but rather as freely floating and readily yielding to the movements of the surrounding organs.

As soon as the uterus is occupied by a fœtus, however, the conditions become changed. In consequence of the increased weight of its contents the uterus exerts tension on the broad ligaments and sinks lower in the abdominal cavity. The vagina and the surrounding connective tissue are rendered tense to a degree depending on the increasing weight of the calf. The uterus then more closely resembles a pendulum, the bob being represented by the fœtus and its envelopes. The suspensory apparatus can be divided into three parts, viz., the two broad ligaments and the tissue connecting the uterus to the vagina.

The pull on the vagina increases greatly as soon as the gravid uterus is twisted either to the right or left, for, torsion being attended with more or less extensive displacement towards the lower abdominal wall, the tension on the vagina must become more marked.

Considering now how the spiral folds and the constrictions which are of such importance in diagnosis are formed, we find that both structures, viz., the wall of the uterus and the ligaments, are implicated, though to different degrees. Whilst the spiral folds are more particularly formed by the wall of the uterus, the broad ligaments are chiefly responsible for the constrictions, though to some extent the spiral folds also contribute to their production. The spiral folds of the body of the uterus are formed solely by twisting of its own walls. This can easily be shown by taking any tubular organ whose walls are not too rigid, and twisting it round its horizontal axis.

The broad ligaments contribute less to the formation of the spiral folds, though they play a more important part in producing constrictions and thus in compressing the wall of the uterus. (Haase, *Berliner Tierärztl. Wochenschr.*, No. 15, 7th April 1904, p. 277.)

AN OUTBREAK OF SARCOPTIC MANGE IN PIGS.

SARCOPTIC mange in pigs has not often been described, so that the following details of an outbreak implicating a large number of animals in one place may prove interesting.

During 1901 M. Scholl saw the pigs in question, and noted incidentally that a large number of them showed considerable irritation and were rubbing themselves on surrounding objects.

At that time he attached little importance to the fact, but on subsequent visits his attention was more closely called to the matter, and he finally concluded that almost all the pigs on this particular farm, including many sucking pigs, were suffering from a pruriginous disease of the skin of a more or less advanced character.

In the meantime many of the animals had seriously suffered in condition. The sucking pigs, though born healthy, fell away greatly in two or three weeks. In a month to six weeks many showed extreme wasting and others died. At this time M. Scholl was asked to undertake treatment.

The first animal examined was a large Yorkshire sow about six years old, which had been directly imported from England five to six years previously. It showed very marked pruritus, and the entire surface of its skin was covered with dry crusts, in some places of a dirty white colour, in others more or less moist.

The more prominent portions of the animal's body were excoriated from rubbing as a consequence of the extreme irritation.

The inside of the ear was coated with a brownish-black, soft deposit, in some places almost an inch thick.

As above stated, this eruptive disease of the skin had affected almost all the animals, but whilst extremely marked in some it was scarcely noticeable in others. M. Scholl diagnosed the condition as sarcoptic mange.

Having been unable to detect anything in a scraping from a young diseased animal, M. Scholl removed some of the crusts from the sow's ear in order to examine them in the laboratory.

To ensure more satisfactory results, he scraped the inner surface of the ear over a considerable area, and mixed the brownish material so obtained with a few cc. of 2 per cent. caustic soda solution. A portion of this material was placed in the incubator for an hour, at the end of which time it appeared converted into a homogenous pultaceous liquid, and was then mixed with water and passed through a centrifugal separator.

The sediment obtained was pressed between two slides and examined under the microscope. This method gave very perfect results, fine examples of sarcoptes in all stages of development being found. The majority were still uninjured and every detail could be noted.

They were evidently Sarcoptes scabei var. suis, the largest variety of Sarcoptes, the female being often half a millimetre in length.

Wishing to further test the action of the soda solution on these parasites, some were allowed to remain for a considerable time in the liquid.

They were still perfect after twenty-four hours' immersion, and only began to break up on the third day. The diagnosis of sarcoptic mange being confirmed, prompt and energetic treatment was evidently necessary, particularly as at least one hundred and sixty animals had to be dealt with.

The measures adopted may roughly be divided into (1) treatment of the animals, and (2) disinfection of the buildings.

The principal building being divided longitudinally into two, one half was emptied and, the litter having been removed and destroyed, the floor, together with the troughs and the walls up to the height of about a yard, were thoroughly cleansed with boiling 5 per cent. carbonate of soda solution. This was followed up by the free use of 5 per cent. sulphate of copper solution. Some days afterwards the walls were lime-washed and the wood-work repainted.

The material chosen for the treatment of the animals was pentasulphide of potassium, dissolved in hot water in the proportion of one kilogramme to thirty litres of water.

Each animal, whether large or small, was first smeared with soft soap and well scrubbed with a brush until the skin was absolutely clean, after which it was freely wetted with the solution of potassium pentasulphide and again vigorously rubbed. The animals thus treated were then passed into the disinfected sties. A dozen which were very severely diseased were dressed with Helmerich's ointment.

From the first day of treatment rapid improvement was noticeable. The irritation diminished, and, as the skin lesions healed, completely disappeared, whilst the animals rapidly put on flesh.

As a precautionary measure, however, the washing and dressing was repeated after the lapse of a fortnight.

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This treatment proved completely successful, for, although two years have passed, the disease has not reappeared in a single animal.

It may be interesting to mention that three very clear cases of transference of the disease from pigs to men were noted during this outbreak. The first was that of the swine-herd, who was suffering from the disease in both fore-arms at the time the pigs were taken in hand. In his case the disease persisted for at least two months. It did not spread beyond the points first noted, but it required careful attention.

The other two cases were in men who carried out the treatment. One was very moderately affected and recovered rapidly, but in the second the disease extended rapidly over almost the whole of the chest and continued for almost three months. Very energetic measures were then adopted and the man recovered.

It seems clearly proved that the Yorkshire sow from which the specimens were obtained had introduced the mischief. The disease more particularly affects well-bred animals. (Scholl, *Ann. de Méd. Vét.*, *May 1904, page 284.*)

ACQUIRED TOLERANCE TO TUBERCULIN.

IN 1892 Nocard stated that tuberculous animals of the bovine species when injected daily or every few days with tuberculin gradually react less and less, and in fact acquire a tolerance of the action of tuberculin. Vallée's observations, however, seem to show that this tolerance is of a very transitory character.

Somewhat later Professor Nocard carried out a series of experiments on this subject. He showed that if a second injection be given twenty-four hours after the first only about one third of the animals so treated react on the second occasion.

The results obtained were little better when the injections were made at intervals of forty-eight hours. Scarcely a third of the tuberculous animals reacted on the second injection.

Even when a week elapsed before the second injection was made only about one half of the diseased animals reacted. After an interval of a fortnight about two thirds of the diseased animals reacted.

"In short," said Nocard, "to obtain a second reaction in all the diseased animals, from twenty-five to thirty days, in round figures a full month, should be allowed to elapse between the two injections."

This tolerance of tuberculin is at the present moment well recognised. Many unscrupulous breeders take advantage of it when selling animals, and thus deceive buyers who take the trouble to test recent purchases before passing them into their premises.

Similarly, at the frontier, where animals which react are rejected, this tolerance of tuberculin is largely utilised by importers, some of whom regularly inject their animals the evening before, or two evenings before, the official test.

The same thing is practised extensively at the different frontiers of Germany; thus, amongst the cattle imported into that country in 1901, 7194 out of a total of 41,808 were found tuberculous, and yet not one of these animals had reacted to tuberculin at the frontier.

It is very desirable that the marvellous diagnostic power of tuberculin should be established beyond doubt, for such incidents, due to misdirected ingenuity, considerably hamper the action of the authorities when dealing with tuberculosis.

In a very remarkable article on the struggle against bovine tuberculosis in Norway, Dr Malm writes: "Errors may occur in the case of animals which have undergone what may be termed a veritable immunisation. But experience shows that this latter is quite irregular, transitory, and uncertain, and that it can be destroyed with relative facility. Such errors, moreover, could be avoided by the adoption of certain administrative regulations; the refusal to supply tuberculin to any persons except veterinary surgeons, the rigorous control of its use, the employment on the frontiers of large doses of very toxic tuberculin, the prolonged retention of animals in quarantine, and the close observation and examination of the animals during the test."

M. Vallée does not question the efficacy of the measures suggested by his colleague, though he considers that many of them are almost impossible to apply, particularly in France.

For instance, it is very difficult to keep imported animals for long in quarantine, whilst it seems impossible to prevent tuberculin being obtained by persons other than those charged with the duty of using it.

MM. Nocard and Roux thought to overcome this difficulty by using at the frontier a special and extremely active tuberculin which produces a prompt reaction in tuberculous animals which have received a large dose of ordinary tuberculin only thirty-six or forty-eight hours previously.

No doubt this material might advantageously have been used, for a time at least, by the veterinary surgeons guarding the frontiers; but the importers would soon have obtained from their own national laboratories a similar tuberculin, and so have endangered the successful working of the French sanitary laws. As the question would therefore again arise *de novo*, Vallée has attempted a fresh solution.

It is first necessary to know the precise nature of the thermic reaction in tuberculous animals subjected to successive injections of one variety of tuberculin. In his experiments Nocard gave several injections of tuberculin, taking the temperature every two or three hours, *but only commencing* after the eleventh or twelfth hour

The question arises whether the first inoculation with tuberculin may not render a tuberculous animal more sensitive, and whether such an animal may not react to the second or third inoculation earlier and for a less time than to the first.

Vallée, using cattle which had just shown a pronounced reaction to tuberculin, gave fresh injections, and by taking the temperature every two hours, *immediately after the inoculation*, was able to detect a rapid and very marked rise in temperature, a rise in every case which was so characteristic as at least to justify one in suspecting the animal. Non-tuberculous animals were unaffected by the treatment.

The results obtained are shown in the following table :—

[TABLE.

No.	First Injection.		Second Injection.	
	Reaction in degrees Centigrade.	Hour at which Reaction was first fully complete.	Reaction in degrees Centigrade.	Hour at which Reaction was first fully complete.
1	2·9	10	2·3	6
2	2·3	11	2·6	6
3	2·7	10	1·7	6
4	2·1	11	2·5	6
5	1·7	11	2·6	2
6	2·7	9	2·1	6
7	2·6	10	1·9	10
8	2·6	11	1·8	7
9	3·1	9	1·8	6
10	2·7	10	3·1	7
11	2·3	10	3·4	9
12	1·8	9	3·6	7
13	1·7	15	3·1	7
14	3·1	11	2·6	5
15	2·1	9	2·1	6
16	2·3	9	2·0	5
17	1·9	11	0·9	8
18	2·0	9	2·3	6
19	2·0	9	1·3	8
20	2·7	9	1·8	5
21	1·3	10	1·0	9
22	1·8	9	1·3	8
23	1·3	9	1·0	8
24	2·1	8	2·7	4
25	1·2	16	1·3	8
26	2·7	9	3·7	4
27	1·3	9	1·6	8
28	2·1	9	1·9	7
29	2·7	11	3·7	3
30	2·2	10	2·1	9
31	1·7	9	1·4	10
32	2·4	8	1·0	10
33	2·1	11	1·1	8
34	2·1	10	1·9	9
35	1·9	10	1·9	10
36	2·4	10	2·2	9

Of a total of thirty-six cattle recognised as tuberculous or suspected of tuberculosis on the first test with tuberculin, not one failed to react to a second test made forty-eight (in some cases only thirty-six) hours after the first, with a double dose of the same tuberculin. The temperatures were carefully taken every two hours immediately after the second injection.

In the first test, carried out under ordinary conditions, thirty-two, of a total of thirty-six cattle, were declared diseased and four suspicious. On the second test, under the above-described conditions, twenty-eight appeared diseased and the remaining eight suspicious.

If this test had been made according to the usual method, in which the temperatures are first taken after the lapse of ten or twelve hours, one would not by Nocard's method have detected a reaction in more than about one-third of the animals; more than twenty would therefore have been declared healthy.

If one studies the curves obtained after the second injection, it is astonishing to note with what rapidity the reaction is obtained.

Thus clear indications were obtained :—

<div style="text-align:center">

At the second hour in one animal.
,,   third·   ,,   one   ,,
,,   fourth   ,,   two animals.
,,   fifth   ,,   three   ,,
,,   sixth   ,,   eight   ,,
,,   seventh   ,,   five   ,,
,,   eighth   ,,   seven   ,,
,,   ninth   ,,   five   ,,
,,   tenth   ,,   four   ,,

</div>

After the eighth hour there only remained therefore nine doubtful results, and at the eleventh hour precise indications had been obtained in all the animals.

If the temperatures had only been taken after the tenth or twelfth hour, as in the case of the first test, the results would have been quite different.

The temperature curve of the second reaction is indicative of a sudden, transitory effect, whilst the initial reaction is always prolonged. Thus No. 14 of the experimental animals gave the following reactions :—

First Injection.   *Second Injection.*

10th hour . .   2·5° C. *4.5°F*   8th hour . .   2·6° C. *4,68°C*

13th hour . .   2·5° C. *4.5°F*   12th hour . .   ·0·8° C. *1,44°F*

17th hour . .   3·1° C. *5.5-5°C*

In the second series of inoculations the temperature after the twelfth hour therefore furnishes little guidance ; the reaction is intense, but occurs very early and is very transitory. Below is a further illustration of the same fact. It represents a secondary reaction :—

First Injection.   *Second Injection.*

6 hours . .   2·7° C. *4.86*   12 hours . .   0·3° C. *.5·4F*

Vallée therefore considers himself justified in concluding, firstly, that in the great majority of cases oxen do not become accustomed to tuberculin ; secondly, that tuberculous cattle almost always react to a second injection of tuberculin given soon after the first, but that this secondary reaction occurs early and lasts a very short time.

Tolerance, if existing, is so little marked that by using animals suffering from pulmonary tuberculosis and observing the above-mentioned precautions, Vallée has obtained an entire series of reactions in the space of a few days.

Below are four examples :—

	1903. 21st Nov.	25th Nov.	27th Nov.
Mammary Tuberculosis	R = 2·9° C. *5:22*	R = 2·3° C. *4.14*	R = 2·6° C. *4.68°F*
	1903. 15th Dec.	16th Dec.	18th Dec.
Pulmonary Tuberculosis	R = 3° C. *5.4*	R = 2·3° C. *4.14*	R = 2° C. *3.6*
	1903. 30th Nov.	2nd Dec.	7th Dec.
Pulmonary Tuberculosis	R = 3·1° C. *5.48*	R = 1·8° C. *3.24*	R = 2·6° C. *4.3*
	1903. 30th Nov.	2nd Dec.	7th Dec.
Pulmonary Tuberculosis	R = 2·1° C. *3.78*	R = 2·7° C. *4.86*	R = 3·8° C. *6.84*

It therefore seems difficult to produce true immunity against the tuberculin reaction. Even when large doses of very toxic tuberculin (fifty grammes of undiluted tuberculin) are employed, one can never be certain of preventing during the following days a reaction from the use of the dose of tuberculin ordinarily given for the purpose of diagnosis.

The above facts suggest a method of preventing the frauds which have now become so frequent in commerce and on the Continental frontiers.

If a veterinary surgeon suspects that a particular animal has been given a dose of tuberculin with the object of defeating the tuberculin test, he should proceed as follows :—

About five or six o'clock in the morning a dose of tuberculin about double that usually employed should be injected.

The temperature should be taken every two hours from the time of injection up to the fourteenth or fifteenth hour.

The intensity of the reaction is indicated by the difference between the temperature at the moment of injection and the highest temperature shown during the following hours.

Any animal which gives a reaction of $1\cdot5°$ C. must be regarded as tuberculous. A reaction of between $0\cdot8°$ C. and $1\cdot5°$ C. should arouse suspicion.

Under no circumstances, however, should the test be made if the animal's temperature is already as high as $39°$ C. The animals should not be allowed to drink during the hour immediately preceding the taking of the temperature. (H. Vallée, *Rev. Gen. de Méd. Vét.*, No. 40, 15th August 1904, page 161.)

THE ULTRA-MICROSCOPE.

HOLMHOLTZ and Abbé stated that, theoretically, the smallest object visible under a dry lens must be at least $\frac{1}{3800}$th of a millimetre in diameter, and in the case of an immersion lens at least $\frac{1}{1800}$th of a millimetre. Siedontopf and Szigmondi, however, in the last volume of Poggendorf's Annals, describe a recently-invented microscope which they term the ultra-microscope. This instrument renders it possible to distinguish surfaces as small as one $\frac{40}{1,000,000}$ths of a square millimetre, or circles with a radius of $\frac{4}{1,000,000}$ths of a millimetre. The ultra-microscope furthermore has the extremely valuable property of facilitating the strongest possible illumination of the object, and thus rendering visible its special peculiarities.

Experiments undertaken in Zeiss' Laboratory in Jena with this instrument have already given astonishing results.

The guiding principle underlying the new method consists in an intense focal lateral illumination, in consequence of which the particles under the microscope reflect the light which falls upon them, and appear self-luminous.

The full significance of the discovery cannot at present be appreciated, but it promises to be of great value for scientific and technical work. (*Kosmos*, 1904, Vol. I., Part I.)

THE INFECTIOUS CHARACTER OF ANÆMIA IN THE HORSE.

AT the present time a very interesting disease of the horse occurs throughout the entire valley of the Meuse, in the neighbouring Departments, and in Normandy. Its principal signs are those of grave progressive anæmia, and it usually ends in the death of the patient. This disease annually kills a large number of horses.

Whilst some authors regard it as due to faulty or insufficient feeding and to bad sanitary conditions, others believe it to be verminous or microbic in character.

As early as 1859 Anginiard reported cases of the transference of anæmia from horse to horse. His statements were confirmed by Ledru, and more recently by Mutelet and Roger (1904). Nevertheless, in 1851 Delafond was un-

successful in his attempts to inoculate horses and sheep with anæmia by injecting them with the blood of diseased patients, and even at the present day the specific organism of the disease is still unrecognised.

Examination of the temperature charts of patients clearly demonstrates the infectious character of the disease, the temperature frequently rising to 40° C. and showing extensive oscillations.

The inoculation of a healthy horse with the blood of certain diseased animals produces rapidly progressive anæmia, absolutely identical with the natural disease.

The disease has thus been successively transmitted through two horses.

The first horse, which was perfectly healthy, was injected into the jugular vein with 750 cc. of defibrinated blood from a diseased animal in the last stage of illness. It developed a typical attack of the disease which lasted fifty-seven days. During this time the animal's weight fell from 472 kilos to 315 kilos. The number of red blood corpuscles progressively decreased from 7,800,000 on the first to 5,700,000 on the fourteenth day, 4,095,000 on the forty-fourth day, 3,505,000 on the fiftieth day, and to 2,280,000 on the day of death. The temperature usually remained in the neighbourhood of 40° centigrade; it rarely fell below 39°, and on the fifteenth day of the disease it rose to 41°.

On *post-mortem* examination the ordinary lesions of anæmia in the horse were found : extreme muscular wasting, subcutaneous, subserous, and perilymphatic œdema, hypertrophy of the spleen, cirrhosis of the liver, and hemorrhages in the bone marrow.

Bacteriological investigations revealed very little. Various microbes, like the colon bacillus and several staphylococci, were isolated from the blood of animals suffering from both the natural and experimental form of the disease, and the same bacteria were found in the viscera immediately after death. They did not appear to play any specific part whatever. The investigators failed to find in the blood either a piroplasma or a trypanosoma, although they tried all the usual experimental methods.

It would therefore appear that the virus of anæmia belongs to the same group as the so-called "invisible" microbes of yellow fever, foot-and-mouth disease, pleuro-pneumonia contagiosa, etc., which are able to pass through filters impenetrable to visible microbes.

They made the following experiments. A mixture of 500 cc. of serum from a patient and 2000 cc. of a dilution in physiological serum of a very rich Pasteurella ovina culture, was filtered through a Berkefeld "candle" a little more porous than the ordinary Berkefeld "V" filter.

The filtrate was opalescent, and when injected into the veins of rabbits and the peritoneal cavities of guinea pigs in doses of 20 cc. produced no bad results. The filter therefore had perfectly retained the very small, virulent microbe of ovine pasteurellosis—it was perfect. 500 cc. of the remaining portion of the filtrate, representing 100 cc. of the original serum from the diseased animal, was injected into the jugular vein of a perfectly healthy horse. After an incubative period of six days this horse showed signs of rapidly progressive and absolutely characteristic anæmia.

It therefore seems proved that anæmia of the horse is an inoculable contagious disease, due to some organism comprised within the group of so-called "invisible" microbes.

This observation suggests also that certain forms of anæmia, like pernicious anæmia of man and the special anæmia of fox-hounds, may also be true infectious diseases.

After inoculation with blood or spleen-pulp from a diseased animal certain subjects contract a benign affection from which they recover spontaneously or which seems in no way to incommode them.

MM. Vallée and Carré are at present studying the properties of the virus of horse-anæmia and the therapeutic qualities of serum from animals "saturated" with virulent blood. (Vallée and Carré, *Rev. Gén. de Méd. Vét.*, No. 39, August 1903, p. 105.)

EQUINE FILARIOSIS.

EQUINE filariosis, recently studied by M. Huguier, is caused by the larvæ of the Filaria irritans Rivolta. It is commonly known under the name of granular dermatitis. In Africa, where M. Huguier lived for several years, he saw numerous cases of the disease, and found that it resisted all forms of treatment and was usually grave in character. Contrary to generally received opinion, it is no commoner in the ass than in the horse.

The disease, which is usually regarded as a simple complication of neglected or indolent wounds, the healing of which has been checked by the heat of summer, is, however, due to an entirely different cause, and is exclusively parasitic and contagious in character.

The method by which the parasites enter the animal's body is not yet fully determined. Huguier adopts Professor Laulanié's theory, that, from eggs introduced into the digestive tract, embryos emerge, which find their way into the blood current, pass throughout the body, and are arrested in the neighbourhood of the wound or in certain organs, where they attain the adult state.

If they invade the skin, the adult worms produce the characteristic lesions of granular dermatitis, but they may also affect the eye, lung, or other organ, the young being carried throughout the entire capillary system.

Having satisfied himself on this point, the author proceeded to study the manner in which animals become infected.

He injected a dog with blood containing embryos taken from a horse, but without producing any apparent infection. He discusses, without, however, admitting, the possibility of the parasites being transmitted by diptera, but, like Laulanié and Nocard, rather inclines to the belief that the eggs of the helminth are absorbed with the food. The disease is not transmissible to man or to the horse by simple contact.

M. Blanchard, who reported on the above observations to the Society of Veterinary Medicine of Paris, does not agree with Huguier's explanation.

He thinks it much more likely that equine filariosis results from the deposition by a dipterous fly of larvæ which attain the adult stage in the skin. The adult worms produce embryos which circulate in the blood, whence they are again taken up by the insect, in whose body they can pass into the larval condition. This cycle of development, which is precisely similar to that undergone by the Filaria Bancrofti in different mosquitoes, is evidently not peculiar to the filaria of man. On the contrary, it represents in general the manner in which a large number of filariæ whose embryos are found in the blood, are propagated. Noé has recently proved that the Filaria labiato-papillosa passes through its larval stage in a very common dipterous fly, the Stomoxys calcitrans, and is inoculated into the ox by the bites of that insect. (*Rév. de Méd. Vét.*, August 1904, p. 469.)

THE TREATMENT OF GANGRENOUS PNEUMONIA BY INTRATRACHEAL INJECTIONS OF CREOSOTE.

THE above method of treating gangrenous pneumonia does not appear to be commonly practised, though it offers great advantages. The following is a short note of two cases in which excellent results were obtained.

On 4th February 1904 Masson and Vazeux saw case No. 1.

All the classical symptoms of advanced gangrenous pneumonia of the right lung were present. The temperature was 40·5° C. The owner stated that the animal, having seemed ill some days before, had been bled and had been given a draught, a portion of which caused it to cough.

The treatment adopted consisted in applying mustard poultices to the chest and administering an electuary containing oil of turpentine, etc. It was suggested to the owner that intratracheal injections should be made. The prognosis was reserved.

On the 5th, 6th, and 7th of February 20 cc. of the following solution was injected :—

Creosote	. . . .	1 part
Alcohol	. . . .	40 parts
Water .	. . . .	40 parts

The injection was made slowly, using a Pravaz syringe and a very fine needle. It produced no attack of coughing, but soon afterwards the discharge became very free.

From the 6th February the discharge lost its offensive odour. On the 7th the general condition had markedly improved.

The temperature was 38·9° C., and the appetite almost normal.

Treatment was then suspended, but during the following days the symptoms became aggravated and the general condition was as alarming as at first.

The temperature rose to 40° C. The number of respirations increased, and the discharge resumed its putrid odour.

Three further injections were made on the 10th, 11th, and 12th, the latter of which produced permanent improvement.

The animal became convalescent and was returned to regular work, at which it has continued since the commencement of March without showing any respiratory difficulty.

Since that time the authors have successfully treated a two-year-old horse also suffering from pneumonia.

Injections were given for six successive days. All other treatment was abandoned from the beginning, and the intratracheal injection of creosote alone relied on. It seems reasonable, therefore, to regard this method as worthy of trial. (*Jour. de Méd. Vét. et de Zoot.*, 31st July 1904, page 407.)

A CASE OF SUPERFECUNDATION IN A MARE.

CASES of superfecundation in the mare are so rare as to deserve being recorded. This is the only reason advanced by M. Emery to justify his communication, for gestation and delivery were perfectly normal.

The mare was a Barb, more than twenty years old, and previously had always been covered by an ass.

On the 20th May 1904 this mare gave birth to a colt and, a few minutes later, to a mule.

When seen by Emery these two young creatures were about a month old, and appeared in excellent health, although a little thin; a fact, however, in no way astonishing, as the mare was very old and herself badly nourished.

The mare's owner had only taken her to the ass, and had no idea at that time that she had been covered by a horse.

It would be interesting to know if the colt, which was born first, had been last conceived, as should have been the case if the rule of primogeniture accepted in human legal medicine were equally applicable to the mare. Unfortunately the facts necessary for settling this point were unattainable. (*Jour. de Méd. Vét. et de Zoot.*, 31st July 1904, page 412.)

TUBERCULOSIS AMONG THE ANIMALS SLAUGH-
TERED IN GLASGOW.[1]

THE following table shows the number of carcases of home and foreign animals slaughtered in the Glasgow abattoirs which were totally or partially destroyed on account of tuberculosis during the years 1901, 1902, and 1903:—

			Cattle.						Swine.	Sheep.	Total.
			Oxen.	Bulls.	Cows.	Heifers.	Calves.	Total.			
HOME	1901	Totals	15	23	918	11	3	970	73	...	1,043
		Partials	35	26	597	6	...	664	1,156	...	1,820
	1902	Totals	33	27	1,128	13	1	1,202	61	...	1,263
		Partials	60	38	749	17	...	864	1,305	...	2,169
	1903	Totals	33	23	1,012	28	4	1,100	75	1	1,176
		Partials	73	38	638	22	...	771	1,905	...	2,676
	1901-3	Totals	81	73	3,058	52	8	3,272	209	1	3,482
		Partials	168	102	1,984	45	...	2,299	4,366	...	6,665
FOREIGN	1901	Partials	2	7	...	...	...	9	...	...	9
	1902	Totals	2	...	1	...	...	3	..	...	3
		Partials	3	4	2	...	...	9	...	...	9
	1903	Totals	1	2	...	...	...	3	..	...	3
		Partials	5	6	...	...	...	11	...	...	11
	1901-3	Totals	3	2	1	...	...	6	...	...	6
		Partials	10	17	2	...	...	29	...	...	29

This Table may appear to show a great pecuniary loss to the trade through the total or partial seizure of so many carcases on account of tuberculosis. These figures are misleading in that they give an exaggerated idea of the severity of the standard by which tubercular carcases are adjudicated, for these Tables were compiled simply to show the prevalence of tuberculosis affecting the lower animals. Many of these animals were consigned to the different abattoirs on account of their unthrifty condition, and might, with equal accuracy, be returned as having been affected with some other morbid condition. To show that many of these animals were in very poor condition, all carcases during the seven consecutive weeks ending 18th June 1904 which were totally destroyed in Moore Street Abattoir on account of tuberculosis were weighed and tabulated as follows:—

From 2nd May to 18th June 1904, there were destroyed, on account of

[1] From the Annual Report of the Veterinary Surgeon (Mr A. M. Trotter), to the Corporation of the City of Glasgow for 1903.

tuberculosis, 150 carcases, weighing a total of 464 cwts., 3 qrs. 22 lbs., or an average of 3 cwts. 0 qrs. 11 lbs. per carcase.

<div style="text-align:center">

5 carcases weighed from 1 to 2 cwts.
83 „   „ 2 to 3 „
*41 „   „ 3 to 4 „
11 „   „ 4 to 5 „
8 „   „ 5 to 6 „
2 „   „ 6 to 7 „
_____
150

</div>

*made up as follows :—

29 carcases, weighing from 3 to 3½ cwts.
12 „   „   3½ to 4 „

Of the 21 carcases weighing 4 cwts. and over—

20 were those of cows, and
1 was that of a bull.
__
21

During the same period the number of cattle brought to Moore Street Abattoir was 3966.

Following the precedent of former years, I herewith submit Tables showing at a glance the prevalence of tuberculosis among (a) the animals reared in this country, and (b) the animals landed at the Foreign Animals Landing Wharf, and the measures taken with those carcases affected :—

<div style="text-align:center">

(a) HOME ANIMALS.

</div>

Species.	Slaughtered.	Tubercular.							
		Affected.		Measures Adopted.					
				Carcases Partially Destroyed.		Carcases Totally Destroyed.		Carcases Passed.	
	No.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Cattle .	47,362	7,484	15·80	771	10·30	1,096	14·64	5,617	75·06
Calves .	2,325	7	·30	...	...	4	57·14	3	42·86
Sheep .	271,385	1	·0003	...	...	1	100·00	..	...
Swine .	53,483	2,132	3·98	...	...	75	3·52	2,054	96·48

This Table is compiled from similar data to that of the previous year.
It differs from the Return showing the total and partial seizures on account

of tuberculosis during the years 1901, 1902, and 1903, in that pig heads are not returned as partial seizures.

(b) FOREIGN ANIMALS.

Species.	Slaugh-tered.	Tubercular.							
		Affected.		Measures Adopted.					
				Carcases Partially. Destroyed.		Carcases Totally Destroyed.		Carcases Passed.	
	No.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Cattle .	39,638	442	1·11	11	2·49	3	·68	428	96·83
Calves .	2	...	...	...	...	...	..	...	..
Sheep .	11,938	...	...	...	...	...	...	...	...

A record of the seat of the tubercular lesions in each animal affected was kept, and are here tabulated :—

HOME ANIMALS.

	Pleural Cavity.	Peritoneal Cavity.	Both Cavities.	Other Parts of the Body.	Total.
Cattle—					
Oxen . .	474	77	275	297	1,123
Bulls . .	512	54	222	180	968
Cows . .	2,081	251	2,657	247	5 236
Heifers .	61	6	66	24	157
	3,128	388	3,220	740	7,484
Calves . .	3	...	4	...	7
Sheep . .	...	...	1	...	1
Goats . .	...		...	...	...
Swine . .	40	109	188	1,796	2,133
	3,171	497	3,413	1,544	9,625

FOREIGN ANIMALS

	Pleural Cavity.	Peritoneal Cavity.	Both Cavities.	Other Parts of the Body.	Total.
Cattle—					
Oxen . .	147	4	32	116	299
Bulls . .	78	4	16	39	137
Cows . .	3	...	...	...	3
Heifers .	. 1	...	1	1	3
	229	8	49	156	442

These Tables are of interest, as they demonstrate that tuberculosis is a preventable disease, and it merely requires stringent hygienic measures, if not to entirely eradicate it, at least to materially diminish its prevalence. If all those cases in which tubercular lesions were found in both cavities be discarded, it will be seen that 88·96 per cent. of affected cattle contracted the disease through the inhalation of tubercle bacilli. This fact cannot be too frequently brought under the notice of stockowners. It has been asserted that only "piners" are dangerous to their fellows, but my experience does not confirm this statement. I have been able, by microscopical examination, to demonstrate innumerable tubercle bacilli in the muco-purulent matter coughed up by an animal in good condition. This animal was, therefore, a "centre" for the dissemination of the seeds of the disease.

The examination (by microscope or by inoculation) of discharges from the lungs and vagina is an invaluable aid to the clinical diagnosis of tuberculosis.

An enquiry into the prevalence of tuberculosis as affecting the different sexes of cattle has been carried out during the past two years. Unfortunately, it was found impossible to differentiate into classes the cattle slaughtered in Moore Street Abattoir. This investigation was, therefore, necessarily restricted to those animals killed in the Milton and Victoria Street Abattoirs.

Percentage of the different classes of home cattle slaughtered in the Milton and Victoria Street Abattoirs found, on *post-mortem* examination, to be affected with tuberculosis :—

1902.

Class.	Number Slaughtered.	Number Tubercular.	Percentage.
Calves	765	3	·39
Oxen	7,902	273	3·45
Bulls	433	50	11·54
Heifers	319	21	6·58
Cows	318	155	48·74

1903.

Class.	Number Slaughtered.	Number Tubercular.	Percentage.
Calves	776	1	·12
Oxen	6,744	267	3·95
Bulls	395	60	15·18
Heifers	320	27	8·43
Cows	199	98	49·24

Percentage of the different classes of colonial and foreign cattle slaughtered in the Yorkhill Foreign Animals Landing Wharf found, on *post-mortem* examination, to be affected with tuberculosis :—

1902.

Class.	Number Slaughtered.	Number Tubercular.	Percentage.
Oxen	24,051	57	·23
Bulls	2,806	36	1·28
Heifers	661	2	·30
Cows	921	32	3·47

1903.

Class.	Number Slaughtered.	Number Tubercular.	Percentage.
Oxen	35,856	299	·83
Bulls	2,826	137	4·84
Heifers	418	3	·71
Cows	540	3	·5

It will be noted that tubercular disease is more frequently found, on *post-mortem* examination, to affect the older home animals, *e.g.*, cows. It ought not, however, to be inferred that aged animals are more liable to contract tuberculosis, but rather regarded as an indication that the conditions under which our herds are housed are favourable to the dissemination of the disease. This view receives confirmation in the statistics regarding the prevalence of tuberculosis among the cattle landed at the Foreign Animals Landing Wharf, York-hill. Of the 921 cows slaughtered during 1902, 32 or 3·47 per cent., were found, on *post-mortem* examination, to be tubercular, whilst during 1903, of 540 cows, only 3 or ·5 per cent., were found thus affected. The disparity in the percentage of tuberculosis affecting home and foreign cows is due solely to the difference of the treatment meted out to these animals. Too many of our herds—even our milch cows—are housed in ill-ventilated, badly lit, and dirty byres. The result is the vitality of the animals is lowered, and a ground specially suitable for the growth of the seeds of tuberculosis is thus prepared. It is, unfortunately, not uncommon to find herds in this country with 50, 60, 70, or more per cent. of their members affected with tuberculosis, and yet the stockowners of this country, with few exceptions, are not taking those measures which have been demonstrated to be effectual to mitigate, if not to eradicate, this scourge.

REPORT OF THE SHEEP-DIPPING COMMITTEE.

THE following are the principal paragraphs of the Report of the Departmental Committee which was appointed by the Board of Agriculture and Fisheries in the month of April 1903 to investigate experimentally, and to report upon certain questions connected with the dipping and treatment of sheep.

Since the commencement of our enquiry an Act of Parliament has been passed relating to sheep-scab, and we desire emphatically to express our approval of the principles contained in the operative clauses of that Act. Its main principle is to give enabling powers to the Board of Agriculture and Fisheries in Great Britain, and to the Department of Agriculture and Technical Instruction for Ireland, to make orders for and to authorise compulsory dipping of sheep.

Preparations for the prevention or destruction of the external parasites of sheep may be classed as dry and fluid. Of these the insecticides applied as a dry powder are of subordinate importance, although one flockmaster stated in evidence that he depended with satisfactory results entirely on this method of treatment.

Fluid preparations may be used in several ways. They may be poured or sprinkled over the sheep, a method, however, that is not now frequently resorted to. In the great majority of cases they are used in a bath, in which the sheep are immersed.

While not wishing to discourage the use of home-made preparations for dipping, we have had sufficient evidence to show that they have to a great extent been displaced by proprietary dips, which in many instances are compounded with considerable skill.

Most of the dips in general use are preparations of arsenic, tar distillates (carbolic acid, etc.), or tobacco, and in arranging for our experiments we selected both home-made and proprietary dips of these types, in addition to the sulphur and lime dip which has been so largely used in some of the Colonies.

With regard to dips, we accept the experimental facts arrived at by Professor Winter. We do not desire to recommend any special dip, as the

evidence and the experiments both lead to the conclusion that the best representative dips of the types commonly used in the country are effective against acari, and, to a great extent, against keds, and are also advantageous for the general health of sheep.

As we are satisfied from the experiments conducted at Madryn that a single dipping, when carefully carried out, is sufficient to cure scab, we have not made any recommendation as to the interval between a first and second dipping, but where, as an additional safeguard, a second dipping is resorted to, we think that the evidence confirms the view previously held that it should take place between ten and fourteen days after the first dipping. Where two dippings are carried out at a less interval than fourteen days, the experiments and evidence prove that arsenical dips should not be used on both occasions.

We are satisfied that the real prevalence of sheep-scab is not adequately shown by the list of recorded outbreaks, as there was undoubted proof, in evidence tendered to us, that in many districts attacks of sheep-scab are not reported.

At the present time the Sheep Scab Orders of 1898 in Great Britain and of 1900 in Ireland are the means by which sheep scab is dealt with, and the powers conferred under these Orders are very wide and can be worked very effectually ; but unfortunately the Local Authorities, who are responsible for the efficient administration of the Orders, are very diverse in their action and in the interest they take in the eradication of the disease.

We have received a considerable amount of evidence, especially from Ireland, which clearly shows that even where the Sheep Scab Order is efficiently worked the penalties inflicted for breaches of the Order are so small that they are valueless as deterrents to the careless flock-owner.

With regard to these Orders we believe that by the use of the powers of inspection conferred upon the Board of Agriculture in Section 2 of the Diseases of Animals Act, 1903, a more effective and zealous administration of their provisions may be secured, and especially with regard to those dealing with isolation of infected or suspected flocks, and the thorough disinfecting of infected premises or folds.

The inspection by the Central Authority should also prove very beneficial in securing an exhaustive examination into the origin of every recorded outbreak of disease, and the actual source of the infection. At present the Local Authority has no power and seldom any desire to trace back the outbreak to any source beyond its own area. In Ireland we are glad to know that the Central Authority has especially called the attention of its inspectors to the importance of this point.

Much dissatisfaction prevails in different parts of the country with regard to the very stringent and diverse restrictions placed upon the movement of sheep into their respective areas by the Local Authorities. These restrictions act in a manner seriously prejudicial to the trade of sheep farming, and in any new regulations which may be put in force to secure the eradication of sheep-scab it is most desirable that uniformity of regulation throughout the country should, as far as possible, be attained, and that some general rules should supersede the narrower local restrictions, which are undoubtedly harassing to trade and vexatious to flock-owners.

The witnesses who came before us were almost unanimously in favour of compulsory dipping. They all recommended that the dipping should be carried out after shearing, when the wool had commenced to grow again, and the majority of them were in favour of immersing every sheep in the tank for a period of one minute.

In our first series of experiments all the sheep were kept in the tank for one minute, but in the experiments dealing with sheep-scab we chose two periods of immersion, in the one case two minutes, the time usually recommended by

manufacturers of dips, and in the other forty-five seconds, a time which more closely approximates to ordinary dipping practice.

In the course of our enquiry we received evidence from New Zealand, and statistics from that and other colonies, showing how sheep-scab has been dealt with. In 1881 it was estimated that there were 700,000 sheep suffering from scab in New Zealand. In 1884 the Government decided to enforce rigorously the statutory powers given them in the Act of 1878, and within two years the scab-infected area was reduced to a small tract of mountainous country. Since 1893 there has been no sheep-scab in New Zealand, and in 1895 a further Act was passed enforcing the compulsory dipping of all sheep within the Colony. The returns from other Colonies are equally satisfactory. In Tasmania practically the entire sheep stock was reported to be infected with scab in 1869, but within ten years after the adoption of compulsory measures the disease was extirpated, and the Colony has been free from sheep-scab since 1879.

After consideration of the evidence from our own flockowners and from the colonies, we have no hesitation in recommending that an annual dipping of all sheep within the United Kingdom should be made obligatory upon all flockowners, and that this dipping should be carried out by the Local Authorities, acting under regulations approved by the Board of Agriculture and Fisheries, and by the Department of Agriculture and Technical Instruction for Ireland. We are fully aware that it is no light task to carry out effectual universal dipping, but we believe that it is the only method of eradicating sheep-scab, whilst its effect generally upon the sheep of the country will be entirely beneficial.

To attain universal dipping, there are two policies which were put before us, one by regulations of the Central or Local Authority, the other by placing the entire responsibility upon the flockowner, and by punishing with ever-increasing penalties every owner for having scab upon his premises, and for failing to cure it. This latter policy undoubtedly proved very effectual in the colonies, especially in New Zealand, but it would involve new legislation, it would be difficult to adapt to the varying conditions of sheep-farming in this country, and it would in our opinion be too drastic a measure to commend itself to flockowners generally at the present time. We have therefore adopted the alternative policy of regulation, and beg to submit the lines upon which such regulations may be framed.

It has been very strongly urged upon us that universal dipping should take place twice a year and not on one occasion only. A double dipping is undoubtedly customary amongst all the most careful flock-masters, but we have not thought it desirable to recommend this at the present time, though we are of opinion that the Central Authority should, under Order, in areas where sheep-scab is firmly established, make special provision for those districts by ordering two dippings within a prescribed period.

To this end we suggest that the Board of Agriculture and Fisheries should establish a schedule of dips recognised as efficient. Makers of proprietary dips who desired to have their preparations placed in this schedule, or users of home-made dips who wished to ascertain whether their compounds would be approved, would then be required to submit samples for examination, for which examination a fee would be charged, and, if satisfactory, the dips would be scheduled as such. To ensure that the dips actually used are not inferior to the samples approved, it should be competent for the inspector to take occasional samples of the dips in use and forward them to the Board of Agriculture for comparison with those originally submitted.

In order to carry out the recommendation that periodical dipping with an effective dip should be made obligatory upon all owners of sheep, we are of opinion :—

(1) That the Board of Agriculture and Fisheries, after consultation with the Local Authorities, should authorise by Order each Local Authority in Great Britain to prescribe regulations by which they shall secure once in each year effective dipping of all sheep (except show sheep and rams which are being prepared for sale, provided they are not permitted to come in contact with other sheep on the holding) by a dip recognised by the Board of Agriculture and Fisheries as efficient.

(2) That the period prescribed for dipping should be arranged for each county by the Local Authority, and should fall between two specified dates within six months after shearing.

(3) That the Board of Agriculture and Fisheries should appoint one or more veterinary or other inspectors for a county or a group of counties for the purpose of seeing to the due execution and enforcement by the Local Authorities of the Diseases of Animals Acts, 1894 to 1903, the Sheep Scab Order of 1898, the proposed Orders above-mentioned, or any future Orders relating to parasitic diseases of sheep, which may be issued by the Board of Agriculture and Fisheries or by the Local Authorities.

(4) That in the event of any Local Authority failing to carry out effectually the above Acts and Orders, that they be administered directly by the Board of Agriculture and Fisheries, the cost of such administration to be charged upon the Local Authority.

(5) That notice of the proposed date of dipping should be given by the owner to the Local Authority, and, after dipping, a declaration should be sent to the Local Authority within a week, stating : —

    (*a*) The number of sheep which have been dipped, and where the same are located.

    (*b*) The day on which such dipping was effected.

    (*c*) The name of the dip or the composition of the dip or material used.

    (*d*) That the dip was properly mixed, and that the dipping was effectually and thoroughly done.

(6) That waste dipping materials shall be disposed of in such a manner as to prevent injury to animals or to any water supply.

(7) That during the period prescribed for dipping no sheep should be removed from any place, farm, premises, or market, except on a declaration from the owner (a copy of which should, immediately after the movement, be forwarded to the nearest police constable) that they have been dipped, provided nevertheless that no declaration should be required for the movement of sheep to a dipping tank for the purpose of being dipped.

With regard to Ireland we recommend that the Department of Agriculture and Technical Instruction should make similar regulations, but with such modifications as the conditions of agriculture in that country may require.

It was represented to the Committee that the carrying out of any scheme of compulsory dipping would be materially simplified by the introduction to the dipping fluid of some substance that would impart a distinct and fairly conspicuous colour to sheep that had been treated according to prescribed regulations. To be practically effective such colouring agent would require to fulfil certain conditions. It would require to be (*a*) obtainable at such a price as not to add materially to the cost of dipping, (*b*) capable of imparting its colour in the presence of various kinds of dips and of impurities in the bath, (*c*) unaffected by the ingredients of dips, (*d*) fairly persistent, (*e*) without effect on the quality of the fleece. The Committee were unable to discover any substance that would satisfy these conditions, and they have therefore no recommendations to make on the subject.

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REPORT OF COMMITTEE FORMED TO CARRY OUT EXPERIMENTS WITH THE VACCINE OF DR PHISALIX FOR THE PREVENTION OF DISTEMPER IN DOGS.

THE idea of preventing distemper in the dog is an old one, dating back to the discoverer of vaccination. Jenner was shown cases of distemper at Berkeley Castle, and on noticing the eruption on thighs and abdomen at once connected it with the variolous diseases of man and cattle to which he was then first giving the attention that has led to such memorable results. Jenner concluded that distemper was the variola of the dog, and advised vaccination as a preventive. Vaccination was, of course, inoculation with the virus of cowpox.

Up to the present time vaccination has continued to be practised, although Dr Walsh, of *The Field*, some thirty years ago experimentally showed that it was ineffective. Believers in vaccination adduce as evidence for their faith the fact that many vaccinated dogs escape the disease, and some relate experiences which are certainly difficult at first sight to explain. Many persons whose credibility must be accepted relate how they have had part of a litter of pups vaccinated with the result that they escaped the disease, whilst the unvaccinated part of the litter all succumbed to it. On the other hand, equally credible witnesses testify to the death of dogs from distemper which had been vaccinated.

The possibilities of infection vary with the circumstances under which dogs are kept, and there are many cases of individual resistance to disease in animals which have been submitted to direct and repeated possibilities of infection. Without an experiment in which the accidental introduction of infection is carefully guarded against and in which the peculiarity of individuals can be rectified by the number of animals escaping or developing disease, no certain value can be given to the preventive agent employed. Dr Walsh and others since have carried out crucial tests demonstrating the uselessness of inoculation with vaccine derived from human or bovine sources.

Modern bacteriology has enabled scientists to produce preventive agents which are active and trustworthy in many diseases—as in rabies, blackquarter, and tetanus. The great demand for a preventive vaccine for distemper has appealed to a number of workers in this department of medicine.

Millais, in 1892, hoped that he had discovered a preventive, but it failed to stand the test of experience.

Dr Copeman—a great authority on vaccination—has also produced a vaccine for distemper, but it has not been successful.

Dr Phisalix, of Paris, is the discoverer of this latest vaccine which has given in some hands good results, and in others has failed. The very contradictory statements of authorities on this vaccine led to public challenges, and finally to the formation of a Committee to carefully test its merits.

This Committee appealed for subscriptions and commenced its enquiry in April 1903. Premises were found in the Wandsworth Bridge Road, Fulham. A well drained, paved, and ventilated building in a large private yard was secured, as it was nearly new and had not been inhabited except by horses since its erection. The process of cleansing was effected, then five separate spaces were enclosed by wood and wire surroundings, so that no direct contact existed between any of them.

First Experiment.

Dogs between two and three months old were advertised for, and were received at various times in litters of from two to five. Each lot came first to Church Street, Chelsea, and was there kept for a few days. As they appeared healthy they were sent on to the experimental kennel and divided between the different wire enclosures. The division was made so that each enclosure contained dogs of about the same age and weight.

A veterinary surgeon—Mr Parsons, M.R.C.V.S.—was engaged to take charge of the whole, and the Committee or some of its members paid weekly visits.

At a meeting of the Committee on 13th May, thirty-three puppies were on the premises, and, all appearing healthy, the 4th of June was fixed for performing the first inoculation.

During this interval, which was given for the purpose of guarding against any of the animals being in the incubative stage of infection, Mr Parsons noticed signs of illness. One dog suffered from intestinal irritation and died. A second soon showed similar symptoms and death rapidly followed. Then one or two were heard coughing, and two days before the day appointed for inoculation catarrhal symptoms appeared in another dog.

On 4th June when the Committee met there was evidence of distemper in some of the dogs in each of the five separate compartments, and it was decided to destroy the whole of the animals in the most humane manner. This was carried out by Mr Parsons, who made a *post-mortem* examination of such as he thought likely to exhibit any interesting lesions.

Second Experiment.

After this unfortunate event the Committee decided to thoroughly cleanse and disinfect the premises, and after a lapse of time to cover the walls of the building with a washable distemper. All utensils and implements were thoroughly cleansed and disinfected.

An additional shed was obtained and five partitions formed in it, so that all litters could be kept distinct for a longer period than before.

Dogs were again sought for, and as they arrived were placed in this new shed to undergo quarantine.

By 25th June we had collected thirty-four dogs of various sizes and breeds, and of ages from two to three months.

On 30th June the Committee met and inspected twenty-four of the dogs which were divided between the five separate compartments in the experimental kennel. As everything appeared satisfactory 13th July was appointed for inoculation of a proportion of the dogs with vaccine No. 1. In the isolation shed a spaniel puppy was found which showed a little extra moisture about the eyes, but no other symptoms.

On 13th July the Committee met and inoculated ten dogs—some from each cage—with 2½ cc. of Phisalix's No. 1 vaccine. All these dogs were in apparent health. There were, however, two dogs—one in cage 4 and one in the quarantine shed—which had refused food and were suffering from gastro-intestinal disturbance. Both died on the 15th.

On 20th July a further batch of ten dogs were inoculated with No. 1 vaccine obtained direct from Dr Phisalix by the kindness of Mr H. Gray.

On 27th July those inoculated on the 13th were inoculated with vaccine No. 2.

On 1st August those inoculated on 20th July with vaccine obtained direct from Dr. Phisalix, were inoculated with No. 2 vaccine.

After this date fatalities occurred as follows :[1]

August 11. Black and tan—found dead (convulsions) 51 V.
  „ 17. Black and white—acute enteritis 56 C.
  „ 26. White terrier—convulsions 78 V.
  „ „ Bull terrier—pneumo-enteritis 64 V.

On 15th August the eight dogs from the quarantine shed were added to those in the experimental kennel, which now contained nineteen vaccinated and twelve unvaccinated, and it was decided to keep the whole with a view to note how the disease which now existed would develop.

This second experiment must be accepted as inconclusive, because although the two puppies which died on 15th July were possibly the victims of distemper infection, it is not certain their illness might not have been due to dietetic causes. By 26th August fatalities due to distemper had undoubtedly occurred, and as we failed to trace the source of infection or the period at which it was introduced it must be allowed that some of the dogs, at the time of inoculation, may have been infected—a condition which renders the preventive action of any vaccine exceedingly uncertain, and destroys any conclusions formed as to the action of the vaccine.

On 9th September a dog suffering from distemper was obtained and allowed to associate with the whole of the experimental animals. Later other cases of disease were also placed in each compartment, with the result that most of the puppies showed some signs of illness, though often only marked by loss of appetite and condition.

The following table shows the number of vaccinated and control dogs with the deaths in each class.

Vaccinated.		*Control.*	
No. of Dog.		No. of Dog.	
51	D. Aug. 11.	53	
52		56	D. Aug. 17
54	D. Sept. 18	76	
55		59	
57		79	
60		69	
61		73	
77		75	
58		80	D. Sept. 12.
62		84	
63		66	
78	D. Aug. 26	67	
68			
70			
71			
74			
64	D. Aug. 26	72 }	D. July 15 be-
65		83 }	fore vaccination.
81			
82			

(D. signifies dead.)

Third Experiment.

After the second failure to ensure the absence of infected dogs it was decided not again to collect a large number of dogs in one building, but to col-

[1] The numbers are those given to each dog. The letter V signifies vaccinated ; C, control or unvaccinated.

lect a few litters and keep each quite isolated until time had shown that no disease existed.

On 28th September a litter of four puppies. two months old, which had been kept after arrival from the country in a loft at Church Street, Chelsea, were taken to the Royal Veterinary College and placed in a loose-box by kind permission of the Principal.

No. 1. A black and white dog
2. ,, ,, ,, bitch
3. ., ,. ., dog with blaze on face
4. A sable bitch.

Nos. 1 and 2 were vaccinated with Phisalix 1st vaccine on the day of admission (28th Sept) and Nos. 3 and 4 were left as controls.

13th October, Nos. 1 and 2. vaccinated with second vaccine.

On 12th October a litter of four Irish terrior puppies about two months old were placed in a loose-box kindly offered by Mr Wm. Sewell, M.R.C.V.S. These were two dogs and two bitches. The latter were kept as controls and the two dogs injected with No. 1 vaccine. On 29th October this litter being healthy and lively the dogs were injected with No. 2 vaccine.

A third litter of four was received from the country and left at Mr Sewell's for the purpose of inoculation after a period of quarantine. In about ten days they showed signs of disease and were poisoned. This litter appeared perfectly healthy on arrival, and their development of the disease was evidence of the wisdom of not bringing together a collection of dogs in which latent infection of one might spread to others and vitiate any conclusions founded upon experimental inoculation. A fourth litter was obtained, but it never got past Church Street, where they all were taken on arrival from the railway station.

On 14th November the litter of four was removed from Mr Sewell's and placed with the four at the Royal Veterinary College. All eight pups appeared perfectly healthy.

20th November. A pup, semi-moribund from distemper, was placed with the eight pups. It was found dead next morning.

25th November. Two black "Chows," both having pronounced symptoms of distemper, were placed in the loose-box with the eight pups. One of the "Chows" was found dead next morning.

Throughout the whole course of the experiments, except when the collie pups were being vaccinated, only the groom who attended to the animals was allowed to enter the loose-box, which was kept locked. The groom had instructions to report to Prof. M'Fadyean as soon as he noticed any indication of illness in any of the pups.

10th December. On this date the groom reported that one of the pups was dull and not feeding. This animal was found to be collie No. 2 (the black and white vaccinated bitch), and on turning it up a distemper papule was found on the inside of one of its thighs. The pup became gradually worse and died on 19th December. The *post-mortem* showed no gross visceral lesions except some small areas of collapse in the lungs.

19th December. On this date collie No. 1 (the black and white vaccinated dog) appeared ill, and it was found to have a characteristic distemper eruption on its thighs and abdomen. A similar eruption was found on collie No. 3 (the unvaccinated black and white dog).

21st December. On this date collie No 1. (vaccinated) died. *Post mortem* as in No. 2.

28th December A similar eruption was found on two of the Irish terriers, one being a dog (vaccinated), and the other a bitch (unvaccinated).

8th January. One of the Irish terriers (a dog, vaccinated) died. During

the previous day or two it had been partially paralysed and showed choreaic symptoms. The *post-mortem* showed no gross lesions, save for some patches of consolidation in the right lung.

25th January. One of the Irish terriers (a bitch, unvaccinated) died. Copious but partially dried-up eruption still visible on thighs and abdomen. No visceral lesions except a small area of consolidation in the left lung.

26th January. On this date collie No. 4 (sable bitch, unvaccinated) was killed. It had been very ill for some days, showing symptoms of chorea, and being unable to stand ; only one distemper papule could be discovered, viz., on the left thigh. The internal organs showed no gross lesions.

There now remained alive the "Chow" introduced on 25th November, and three of the experimental puppies, viz. :—

Collie No. 3 (black and white dog with blaze on face, unvaccinated). It had now apparently quite recovered, and the cutaneous eruption had disappeared.

An Irish terrier dog (vaccinated).

An Irish terrier bitch (unvaccinated).

Neither of these Irish terriers had ever shown any decided symptoms of illness, and both now appeared well.

To bring the experiments to a close these four animals were destroyed on 26th January.

Conclusions.

The Committee consider the first experiment an entire failure, and the second inconclusive but suggestive.

On the other hand, they regard the results of the third experiment as unimpeachable evidence that the vaccination failed to confer any immunity against distemper. The first two animals to die from distemper were the two vaccinated collies ; and while both the control collies contracted the disease, one of them recovered. In the case of the Irish terriers, one vaccinated and one unvaccinated succumbed to the disease, but the first of the two to die was a vaccinated pup.

The net result of the experiment thus was that three of the four vaccinated pups died from distemper, while only two deaths occurred among the four unvaccinated pups.

Mr Parsons' Report.

The animals were kept in a light, lofty, airy stable with granite floor and good drainage. The entrance faced south, and on cold windy days the place was inclined to be draughty.

There were five cages made of wood and wire netting, and so constructed that an occupant of one was unable to come in contact with one in another cage. Size of cages varied from 12 ft. by 12 ft. downwards.

The floor of the cages was kept covered with sawdust, and shavings were used as bedding. Each cage was furnished with its own feeding utensils, which were never interchanged.

The puppies were fed three times a day and received such quantities and mixtures of food as seemed desirable to their age and size. Horseflesh, bread, biscuits, and milk were used. The flesh was cooked on the premises and mixed whilst meat and broth were hot, being served when cool.

No dogs were placed in the experimental kennels until after a quarantine in another shed of at least ten days.

Skin Eruption.—A large proportion suffered at some time during their stay from red spots or vesicles on their bare abdominal skin and on the bare skin inside thighs. These eruptions were commonly three or four in number. They recurred in some instances, the interval between these eruptions varying from two or three days to three months (77). They were very superficial,

and usually lasted about twenty-four hours. All traces of these eruptions disappeared usually within a week.

The eruptions varied in size, being commonly from one-eighth inch in diameter, more rarely a quarter inch in diameter. They were commonly circular, but occasionally oval or irregular. They were usually of a yellow or pink colour; more rarely they were white with purulent contents. In certain instances a red areola surrounded the vesicle or pustule, but this was exceptional. In one instance an exanthema was seen a week before pustules. The clear yellow vesicles did not alter their character—they did not become pustular.

In some instances, about a week or two after these eruptions had subsided, there appeared a number of dark brown spots around site of vesicle, varying in size from that of a pin-head downwards.

Pulmonary.—In seventeen out of twenty-two some lesion of bronchopneumonia was discovered on *post-mortem* examination.

The symptoms of this condition during life were not marked. In a small proportion a slight cough was noticed on certain occasions. The most common and persistent symptom in these cases was slowly progressive emaciation.

Gastric and enteric.—Enteritis in varying degrees was met with in many instances.

In one or two cases there were numerous ulcerated areas and intestinal contents were blood stained, but this latter appearance was also seen in the absence of ulceration when the enteritis was acute.

In many cases the enteritis appeared in small patches throughout the whole length of the intestine.

Gastritis was met with in fewer instances, and usually large areas were affected.

The symptoms were usually limited to diarrhœa, dysentery was observed in a few instances, vomition was of rather rare occurrence. In some cases none of these symptoms appeared.

Catarrhal.—Congestion of conjunctivæ was of common occurrence. The condition frequently recurred after periods of subsidence of varied length.

Serous discharge was also common in presence or absence of the above condition.

Corneal ulceration or cloudiness was seen in four or five cases.

Mucous nasal discharge was seen in some instances, serous discharge was more common.

Ecchymoses and other lesions.—In some instances sub-endocardial, subperitoneal, and sub-pleural ecchymoses were discovered, and small hæmorrhages into the substance of the cardiac muscle were occasionally met with.

In one case numerous small hepatic abscesses were seen.

One animal—a spaniel—suffered from arthritis, the hock-joint becoming distended with fluid.

Temperature. — There was nothing typical in the rise and fall, each individual case differing in this respect.

In some instances death took place in an animal whose temperature had never exceeded 103°.

In others there was high temperature for one, two, on more days, or one or more occasions spread over periods of three or twelve weeks.

In a few, periods of high temperature recurred at intervals for weeks, and in one case for three months.

Nervous.—Nervous symptoms were noticed in one or two instances. One case showed skin eruptions on 8th July and subsequently. Shivering, conjunctival congestion, and yellow coagulated eye discharge on 19th and 20th July.

Mucous eye discharge 25th July, coughing 26th August and subsequently, and dulness, temperature 104·4°, and a number of epileptic seizures on 30th August. Emaciation was noticed to commence about this date. The animal was still living on 3rd October.

In another instance one died two days after an epileptic seizure. *Post-mortem* : broncho-pneumonia, enteritis, and ecchymoses (sub-endocardial). In no case was paralysis or chorea noticed.

The Vaccinal Reactions.—1. *General.* As a rule no general reaction followed vaccination.

In one instance slight dulness was noticed on the second day after the first vaccine, and one yellow vesicle appeared on the seventh day.

This animal on the third day after the second vaccination suffered from slight loss of appetite, and on the fourth day from congestion of conjunctivæ, and died on the fifteenth day suddenly after showing very slight dulness, congestion of conjunctivæ, serous eye discharge for a few days, and temperature of 104·4° on day before death. Found dead with mouth full of food.

To First Vaccine.—2. *Local.* In three cases no reaction.

In two cases redness on following day or two on vaccination area.

In three cases redness and slight swelling.

In two cases very slight swelling only.

In nine cases circular swelling, consistence of lymphatic gland, of ½ to 1 inch in diameter, and raised from ¼ to ½ inch.

In one case (54) there was a prominent swelling measuring 1½ inch by ¾ inch by ¾ inch on third day after vaccination. This animal subsequently developed nasal catarrhal symptoms.

To Second Vaccination.—In four cases there was no reaction.

In seven cases there was merely a swelling along course of needle track not much more than ⅛th inch in depth.

In one case there was redness only.

In eight cases circular swelling from 1½ to ¼ inch diameter, and ½ to ¼ inch in depth.

ROYAL VETERINARY COLLEGE, LONDON.

LIST OF BURSARIES, MEDALS, HONOUR CERTIFICATES, ETC., 1904-1905.

Coleman Prizes.

Silver Medal	Mr T. C. Graves.
Bronze Medal	Mr W. Urquhart.
Certificate of Merit	Mr R. L. Phillips.

Centenary Prizes (£20 each).

Class A	Mr B. Hughes.
Class B	Mr A L. Sheather.
Class C	Mr J. Godber.
Class D	Mr T. C. Graves.

Royal Agricultural Society's Medals.

Silver Medal	Mr R. L. Phillips.
Bronze Medal	Mr R. Bennett.

Ralli Prizes in Practical Surgery.

First Prize £5 5s.	Mr H. C. Dibben.
Second Prize £3 3s.	Mr R. L. Phillips.
Third Prize £2 2s.	Mr W. J. Moody.

Clinical Prizes.

Class A.

First Prize £5 . . .	Mr G. Yates.
Second Prize £3	Mr W. D. Halfhead.
Third Prize £2	Mr H. A. Thorne.

Class B.

First Prize £5	Mr T. Lishman.
Second Prize £3	Mr W. G. Blackwell.
Third Prize £2	Mr H. Bone.

Class C.

First Prize £5	Mr H. A. Lake.
Second Prize £3	Mr W. T. M. Browne.
Third Prize £2	Mr F. Chamberlain.

Class D.

First Prize £5	Mr L. E. W. Bevan.
Second Prize £3	Mr R. Bennett.
Third Prize £2	Mr W. J. Moody.

CLASS PRIZES

Class D.

MEDICINE.—*First Prize*—R. L. Phillips. *Second Prize*—T. C. Graves. *First-class Honour Certificates*—R. Bennett, J. Blakeway, L. E. W. Bevan, E. Browne, T. Bone, W. T. Collins, E. E. Elphick, T. C. Graves, J. R. Hodgkins, R. L. Phillips, W. Trigger, W. Urquhart. *Second-class Honour Certificates*—J. J. Aveston, H. C. Dibben, W. J. Dale, W. J. Moody, D. Quinlan, F. V. Steward, R. Tindle, E. R. H. Woodcock, A. Whitehead.

SURGERY.—*First Prize*—T. C. Graves. *Second Prize*—L. E. W. Bevan. *First-class Honour Certificates*—L. E. W. Bevan, T. C. Graves. *Second-class Honour Certificates*—J. J. Aveston, R. Bennett, J. Blakeway, E. Brown, T. Bone, W. J. Dale, E. E. Elphick, J. R. Hodgkins, W. J. Moody, R. L. Phillips, W. Trigger, W. Urquhart.

Class C.

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HYGIENE.—*First Prize*—J. Godber. *Second Prize*—L. Danels. *First-class Honour Certificates*—L. Danels, J. Godber. *Second-class Honour Certificates*—W. A. Broad, R. Branford, E. Belcher, W. T. Brookes, F. W. Chamberlain, A. Douglas. C. C. Parsons, W. H. Simpson, W. H. Thomas, W. H. Taylor, H. S. Ward.

MATERIA MEDICA.—*First Prize*—J. Godber. *Second Prize*—L. Danels. *First-class Honour Certificates*—L. Danels, J. Godber. *Second-class Honour Certificates*—R. Branford, E. Belcher, F. W. Chamberlain, A Douglas, H. A. Lake, C. C. Parsons, G. G. Sooby, W. H. Simpson, W. H. Thomas, W. H. Taylor, H. S. Ward.

Class B.

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HISTOLOGY. — *First Prize*- M. Henry. *Second Prize*—A. L. Sheather. *Second-class Honour Certificates* — J. R. Crane, L. A. F. Dawson, P. L. Edward, M. Henry, F. E. Jones, M. H. Kingcome, T. Lishman, M. Morphew, A. W. N. Pillers, G. B. C. Rees-Mogg, A. L. Sheather, W. R. B. Wakeham.

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Class A.

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PRACTICAL CHEMISTRY.—*First Prize*—H. Tudor Hughes. *Second Prize*—D. O'Kell.

BIOLOGY.—*First Prize*—B. Hughes. *Second Prize*—H. Tudor Hughes. *First-class Honour Certificates*—J. Hadfield, B. Hughes, H. T. Hughes, G. Tillyard, W. Waters. *Second-class Honour Certificates*—H. J. Allen, E. Brogan, A. Bowhay, H. M. Holland, W. Jones, P. S. Morgan.

MINOR ANATOMY.—*First Prize*—W. Waters. *Second Prize*—E. H. Brogan. *First-class Honour Certificates*—E. H. Brogan, J. Hadfield, H. M. Holland, W. Waters. *Second-class Honour Certificates*—H. J. Allen, A. Bowhay, C. M. Barton, W. F. Bright, D. Chatterley, A. Comerford, W. D. Halfhead, B. Hughes, H. T. Hughes, W. Jones, S. J. Motton, D. O'Kell, G. Tillyard, H. Tay.

PASS LIST.

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SECOND PROFESSIONAL EXAMINATION.

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[1] In this and the succeeding Pass Lists † indicates with First-Class Honours, and ' with Second-Class Honours.

W. P. R Beal, E. Blamey, H. Bone, S. Carless, J. R. Crane, *L. A. F. Dawson, P. L. Edwards, F. B. Greer, R. L. L. Hart, M. Henry, C. Hartley, E. V. Hobbs, T. E. Jones, M. H. Kingcome, *P. Kerr, V. C. Leckie, T. Lishman, H. Morphew, *A. W. M. Pillers, G. B. C. Rees-Mogg, E. D. Sewell, Sidney Smith, *A. L. Sheather, A. E. Webber, W. R. B. Wakeham, T. Ware, J. S. Young.

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‡ Receives diploma 26th Nov. 1904. § Three years' course.

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THIRD PROFESSIONAL EXAMINATION.

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FINAL EXAMINATION.

Messrs R. W. Jones, W. D. Lindsay, J. C. Smith, F. R. Shippard, J. B. Chadwick, H. E. Cross, J. A. Fearnside, *R. Mairs, P. Reilly, K. P. Rankin.

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SECOND PROFESSIONAL EXAMINATION.

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THIRD PROFESSIONAL EXAMINATION.

Messrs *J. T. Clinton, J. J. Fleuery, Thomas Flynn, S. J. Richardson.
Class C (Three Years' Course)--P. J. X. Kearney, Veterinary Department, Bulawayo.

FINAL EXAMINATION.

Messrs W. J. B. Devine, *A. Baker, A. C. Duncan, Jos. Lynch, R. G. Melville, *G. W. Tyson, E. A. White, M. J. Herriott.

THE EVOLUTION OF IMMUNITY.

By GERALD R. LEIGHTON, M.D., F.R.S.E., Professor of Pathology,
Royal (Dick) Veterinary College, Edinburgh.

MY object, in this paper, is to endeavour to suggest some thoughts
which force themselves upon us when we set ourselves to enquire
into the problem of the origin of the phenomenon known as immunity,
and first of all let us be perfectly clear as to the meaning of that
term.

When we look carefully at the various species of highly organised
animals with a view to ascertaining their specific differences, we find
that it is not in structure alone that one species of animal differs from
another.   There are other characteristics no less striking that separate
species, characters which are doubtless connected with their ultimate
material structure, but which it is impossible in the present state of
our knowledge to correlate with specific morphological variations.
Thus we find that almost all vertebrate species of animals exhibit
differences in their liability to diseases, differences either of degree
or kind or of both.   All vertebrate species have their own special
mental qualities, exhibited by varying degrees of intelligence
and acuteness of sense perceptions.   Some appear to be possessed
of almost human reason, at any rate they are extremely sus-
ceptible to educative influences, whilst others resist these influences
almost entirely.   In a word, specific differences embrace every
possible variation in physiological activities, whether these be what
we term mental, moral, or physical.   Taking one only of these classes
of variations, we may from that arrive at a definition of immunity
which, with but slight verbal alteration, would be applicable to all.

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It is convenient to take the physical differences as indicated in the power of resistance to disease, because this special aspect of immunity (and it is only one aspect) is of most importance to us as scientific students.

Two or three well-known examples will suffice.

Horses are very susceptible to glanders, it is the most fatal equine disease ; horned cattle are immune to glanders ; goats and sheep are intermediate. Some horses are more susceptible than others. Cattle and sheep are very liable to anthrax ; but Algerian sheep are almost immune to anthrax, and horses are not nearly as susceptible as cattle.

Tuberculosis, the most wide-spread human disease, exhibits great variation in its attacks upon animals. Cattle are most susceptible, while it is far less common in sheep and goats. Field mice are susceptible to tubercle, whilst house mice and white mice are resistant. Finally, man suffers from many conditions whlch do not attack the lower animals, and *vice versa*.

In the light of these facts relating to the varying incidence of disease, it is not difficult to frame a definition of immunity sufficient for our purpose. We may say that : " Immunity is that condition of an organism or tissue in virtue of which it is able to resist infection either partially or absolutely."

We are not concerned just now with the exact nature of this condition ; that is a complicated question which it is not my purpose to deal with now. The question I wish to ask is : Can we account in any degree for the origin of the phenomenon, or for the differences shown by animals in relation to it ? In other words, what can we discover as to the course of the evolution of immunity, and how have animals become immune ?

It is only necessary to state such a problem in order that its enormous importance should be realised. The possibility of becoming immune to destructive agencies and deleterious influences of any and every kind, is the greatest hope of mankind, and there can be no more interesting and important enquiry than that of how specific immunity has been evolved in the past, and how its evolution is progressing in the present.

When we come to look a little closer at the manifestation of this phenomenon of immunity, we at once discover that it presents itself in two perfectly distinct forms. It is seen that there are two forms of immunity to disease. For example, some species exhibit an innate immunity to certain infections, such as horned cattle to glanders, as above-mentioned. The immunity is inborn in the species. But there is another form of immunity which is not inborn but acquired, as an example of which may be mentioned the immunity of human beings to such diseases as smallpox, measles, and scarlet fever, after recovery from an attack of these conditions. This immunity is acquired naturally by the individual who recovers. (There is also an acquired immunity, the result of specific treatment by inoculation, but this we have not to consider now).

Immunity is therefore (*a*) innate, or (*b*) acquired, and the problem to which I wish to draw attention now is first, how is innate immunity evolved ? and second, how is the power of acquiring immunity evolved ?

It will simplify matters if we restrict ourselves to the evolution of immunity to disease in human beings, because we have more facts on this aspect of immunity than any other, and we may be quite certain that the same laws have produced immunities to other influences.

We may select smallpox, tuberculosis, and measles, as well-known diseases to investigate, and our own race, the Anglo-Saxon, as a case in point.

It will at once be evident that smallpox and measles are on a different footing to tuberculosis, for, whereas the patient who recovers from an attack of the two former is relatively or absolutely immune from future infection, the exact opposite is the case in tuberculosis—one attack renders the patient more liable to a second.

As the case of smallpox is complicated in this country by the prevalence of vaccination, I will leave that for the present and consider the other two, measles and tuberculosis, against which we have no specific treatment as yet, and whose effects on a race may therefore be traced as those of any other factor which contributes to the evolution of any race.

Measles may be said to be endemic in most civilised countries, though by no means equally common. " It is liable to some epidemic extensions, the recurrence of which in European communities is characterised by striking periodicity. In isolated communities the disease will occasionally die out altogether, but after the lapse of some years, during which a susceptible population has been growing up, the importation of a fresh case is liable to result in a fresh outburst which may rapidly assume pandemic proportions. This was well illustrated in the case of the Faroe Islands, into which, after an immunity from measles lasting sixty-five years, the disease was re-imported in the year 1846, with the result that in the course of five months more than three-fourths of the entire population were attached ; and the same thing in even more marked degree was seen in Fiji in the year 1875, when the disease was imported into the country, it is supposed, for the first time. In this instance, so extensive was the outbreak, and so rapid was its spread, that more than one-fourth of the population actually died of the disease within three months."[1]

Such a catastrophe as that which occurred in Fiji is nowadays quite unknown in Great Britain although measles is constantly with us. It is here considered, apart from its sequelæ, a trifling complaint, which most children recover from with no further treatment than any careful mother may carry out. And yet, recollect, it is extremely common. What do these facts mean, but that our race has become immune, not to measles. but to the danger from measles ; in other words we have evolved the power of acquiring immunity to measles, a very different thing from evolving immunity to the disease itself, and a thing which obviously neither the inhabitants of Faroe or Fiji had evolved. Introduced into a new locality for the first time, measles is seen to be an extremely fatal condition. If that locality be isolated the inhabitants become immune for a time, and then again susceptible, while in a large community such as ours what takes place is that in course of time the race is able to survive the disease, although it never becomes immune to the infection. Coming amongst the Fijians, one-

[1] " Encyclopædia Medica," Vol. VII., pp. 348, 349.

fourth of the population died forthwith, because they had not had time to evolve the capacity for acquiring immunity against it.

So that, whatever the exact explanation may be, which is the problem before us, it is very evident that the result of long continued exposure to the ravages of measles has resulted in the evolution amongst Englishmen of a greatly increased power of resistance to the effects of measles, a power, in other words, of acquiring an individual immunity, without which evolved power a race exhibits a great mortality from this infection. It is quite evident, also, that we shall never evolve immunity against the infection, but that as time goes on we shall exhibit an increased capacity to resist the effects, that is an increased power of acquiring immunity.

The history of smallpox is from the point of view of race evolution on similar lines.

" Smallpox is known to have been indigenous in Eastern countries for nearly two thousand years, its earliest records coming from India and China. The first appearance in Europe seems to have been in the sixth century, when fatal epidemics broke out in southern France and northern Italy. England appears to have been first severely visited about 1241, and at this time the disease was widely spread through Europe. From Europe smallpox was conveyed to the American continent, first to Mexico in the year 1527, where it raged to a fearful extent, destroying millions of victims, and spreading thence to other parts of America, which it gradually overran."

" Smallpox has been one of the greatest, if not actually the greatest, of the scourges of mankind ; no form of disease has been more destructive of life, none has brought more misery in its train. In Europe not a decade passed in which the disease did not overrun great tracts of country, decimating the inhabitants in its progress. In England in the seventeenth and eighteenth centuries a large proportion of the medical literature bears upon the treatment of smallpox, showing that that disease was the common form of illness which the physicians of those days were called upon to treat." Nowadays in this country one may be many years in practice without seeing a single case.

" The records of the mortality from smallpox in the eighteenth century show that it accounted for nearly one-tenth of the total mortality, that neither age nor sex nor social conditions afforded protection, and that of those who recovered from it the number permanently scarred or disfigured formed so large a proportion of the population that unmarked persons were comparatively few." Indeed, to be unmarked constituted in a woman a claim to be considered a beauty.[1]

At the present time in European countries smallpox has been robbed of much of its terror owing to the introduction of vaccination, which is an artificially acquired immunity, but, even apart from vaccination, smallpox is not the scourge it was formerly, that is to say, the death rate from it is not so high in those attacked. The population remains susceptible to its infection, but has evolved the power of resisting its effects to some extent, so that a greater proportion of cases recover. Just as in the case of measles, so in smallpox, there is no sign of any evolution of an innate immunity,

[1] " Euclyopædia Medica," Vol. II., pp. 213, 214.

people are as naturally susceptible as ever; indeed, susceptibility is almost universal, but, as in measles, where a nation has been exposed to its ravages for many generations, they gradually evolve the power of acquiring immunity in individuals, and so reducing the death-rate percentage of cases.

What about those races into which smallpox is introduced for the first time? History has no more striking records than those relating to this matter. The experiment has been carried out upon such a colossal scale, and with such dire results to the nations, that he who runs may read. As we have said, they are literally decimated when first exposed to smallpox. Indeed, so powerful is the smallpox poison in a new blood that even the attenuated virus of vaccination is more than a nation unused to smallpox can stand, and, as in the case of the Esquimaux, they will die from it. We, who have had smallpox among us for centuries, are evolving a capacity to recover from infection, a capacity which enables us to make use of vaccination as a safeguard, because that attenuated virus causes us as a rule no more than a temporary and slight discomfort, while at the same time it confers an immunity which lasts for some years. Our resisting power to smallpox is not yet on a par with that to measles, but it is proceeding upon precisely similar lines, and for precisely the same reasons. What those are we shall see presently.

But now consider the case of tuberculosis for a moment. Tuberculosis is the most widespread of all human diseases, and the majority of the higher vertebrates are also susceptible. It has been said that 7 per cent. of mankind die from its effects. It is due to an infective organism, and in its most common form—consumption—the disease "for very many centuries has ravaged the Old World, especially such crowded parts of it as England. But Englishmen now increase and multiply in cities and towns, the natural breeding places of consumption; whereas, under like conditions the inhabitants of the New World, where consumption was unknown till recently, perish. When infected with consumption by white men they tend to extinction everywhere, even in rural districts." Whole tribes of native races have perished as the result of their first contact with this white man's scourge. But the white man crowds himself in millions into the slums of London, Manchester, Glasgow, Edinburgh, and numbers of other large cities, and still increases and multiplies.

Now note: one attack of consumption does not confer a lifelong immunity upon the patient, as is commonly the case in measles and smallpox; on the contrary, one attack of any form of tuberculosis leaves the patient more susceptible to the disease than he was before. In order that he may have a chance of survival, he must be taken out of infectious surroundings and tended with the utmost care. The patient recovered from smallpox can face an epidemic with impunity, but the tubercular patient who has recovered must avoid the source of the evil. How then is it possible for Englishmen to increase and multiply under the conditions of our slums which are so favourable to tuberculosis? Obviously the only possible answer is that a great evolution against tuberculosis has occurred, but in this case what has been evolved is an inborn or innate immunity. No increased power of resistance is to be found; the explanation is that the infection itself is resisted. The race does not recover from the infection, as it does

from measles; it resists it. In the one case the capacity to recover, the power of acquiring immunity, has been evolved; in the other case the capacity to resist, an innate immunity, has been evolved.

Such then are the facts of the case. What is the argument of the problem? How have these results come about? How is immunity evolved?

It is quite clear that it would be ridiculous to place this problem outside the scope of any other phase of physical evolution. The explanations of the evolution of one part of man's nature are to be applied to all parts. Those explanations during the past hundred years have centred themselves round two main views, the Lamarckian and the Darwinian, and it becomes of great interest from the point of view of the general question of evolution to see which theory is capable of affording an explanation of the evolving of immunity.

"Only two theories of evolution are possible, or even thinkable. All other theories, of which several have been enunciated, are from the nature of the case, mere variations of those two."

" Evolution must proceed by the transmission of inborn traits, or of acquired traits, from parent to child, and by their accentuation during succeeding generations. Even if we regard the course of evolution as designed and predestined, no other method is thinkable, because no other traits than the inborn and the acquired exist in living beings." " Lamarck, because he first formally enunciated it, gave his name to the one theory; Darwin, for the same reason, gave his to the other." [1]

Lamarck held that characters which have been acquired during the life of the parent are transmissible to some extent to the offspring, and that the accentuation of these transmitted traits during successive generations was responsible for evolution. In other words, the Lamarckian view is " that variations arise through the transmission of parental acquirements to the child, and that evolution results from the repetition of this process during succeeding generations." [2]

Darwin, although he thought that some acquirements were transmissible, perceived that this explanation was utterly inadequate for all the facts, and insisted that variations in offspring occurred quite apart from any previous acquirement by the parent; that they arose independently of any known law, and hence are termed spontaneous variations. He further insisted that evolution was mainly due to the selection by Nature of those variations which gave their possessors some advantage in the struggle for existence, and so it comes about that Darwinism is summed up in the phrase, " The survival of the fittest by Natural Selection in the Struggle for Existence."

The modern followers of Darwin go further still, and assert that Lamarck was entirely wrong; that acquired characters are never transmitted, and that Darwinism, therefore, is the whole solution of evolution. " It is curious that during every age men have accepted as a belief the method of evolution as laid down by Lamarck, but have sought to improve their own plants and animals in the manner described by Darwin." [2] That is to say that breeders select well-marked spontaneous variations to breed from, not animals which acquire new characters in their adult life.

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[1] "Alcoholism."—G. Archdall Reid, pp. 7, 8.   [2] Ibid., p. 8.

Now apply these two views of the method of evolution to the phenomenon of immunity.

According to Lamarck, characters acquired by the parents are transmissible to the offspring. Disease is an acquirement; so is immunity to disease after recovery from an attack. We should expect on this view that diseases against which the individual cannot acquire immunity would be transmitted, whilst in the case of the parent becoming immune that immunity ought to be transmitted likewise. It is at once apparent that the facts of the two diseases we have considered, measles and smallpox, do not accord with these expectations. Generation after generation of parents acquire a life-long immunity to measles as the result of an attack, and yet the children of these parents are born still susceptible to the infection. They are just as liable to measles as were their parents who acquired immunity. The children in their turn have to acquire their own immunity. The same is true of smallpox. True, the succeeding generations of offspring exhibit an increasing power of recovery from these diseases, but that cannot be explained by the view of Lamarck. On the other hand, if a parent acquires tuberculosis, his children on this view should be born with tuberculosis, which they are not. If tuberculosis were transmissible from parent to offspring, it is quite obvious that in each succeeding generation there would be more and more consumptives, and the race would progressively degenerate. We have seen that as a matter of fact the precise opposite is the truth; that the longer the race is exposed to the disease, the less severe is the result, the fewer the deaths. Immunity to tuberculosis could not be evolved on the Lamarckian hypothesis, because the individual does not acquire it to transmit.

Lamarckianism, then, offers no explanation of the evolution of immunity, nor does it explain the origin of the power of acquiring immunity.

Turning now to Darwinism, the doctrine of the survival of the fittest in the struggle for existence, we are at once impressed with the thought that, if this doctrine be true, disease must be a potent factor in the evolution of man, for it is the most ruthless of all selecting agencies. In the struggle for life against infectious disease, other things being equal, it is the fittest which survive, and the fittest in this case are those individuals who are naturally immune, or who possess the greatest capacity of acquiring immunity. In the case of measles and smallpox, out of five hundred persons attacked a certain number will be able to recover from the disease, that is, will be able to acquire immunity against it. We see that their acquired immunity is not transmitted, their children also take measles and smallpox; but the power of acquiring immunity, because inborn, is transmitted to the offspring, and, inasmuch as the parents are selected, the power of acquiring immunity tends to increase in succeeding generations. The most susceptible die out, leaving the least susceptible to carry on the race, and this selection of the fittest acting over many generations will gradually result in the evolution of an increased power of recovery, of acquiring immunity. Hence, the longer measles and smallpox and other similar diseases act upon a population, the more will the unfit be eliminated and the less susceptible will successive generations be. Hence the general law that "Every race is resistant

to every deadly disease strictly in proportion to its past experience of it."[1]   Hence Englishmen can live and multiply although constantly exposed to measles and tubercle, where whole tribes are wiped out on their first introduction to these infections.

In the case of tuberculosis, a disease against which the individual acquires no immunity, what should we expect on the principle of the survival of the fittest?   Obviously that only those who possess an inborn immunity will survive, and that the most susceptible will perish.   The inborn immunity is transmitted to offspring, and, on account of the elimination of those who have it not, in each succeeding generation there will be a greater proportion of immune individuals. They are selected by nature to carry on the race, which for this reason gradually evolves an inborn immunity.   Hence, numbers of Englishmen can live and multiply in badly lighted and ill-ventilated slums, although constantly exposed to the tubercle bacillus, whilst the native, even in the open wilds, is carried off in large numbers.   "These diseases are so prevalent within their areas of distribution that no man escapes infection unless he be immune, nor death unless he be resistant."

Plainly there has been great evolution against disease, but also plainly it has not been by the transmission of acquirements, but by the elimination of the unfit by natural selection.

The conclusion we are inevitably driven to, therefore, from a study of the past history of the relations of man to infectious disease, is, that both innate immunity and the power of acquiring immunity are evolved in the same manner as are other characters, namely, by natural selection of the fittest to survive.

" If the effects of disease are transmissible, then their effects must accumulate generation after generation.   The son must start with the parent's constitution plus the effect of the parent's disease, the grandson   must   start   with   the   son's   constitution,   plus   the effect   of   the   son's   disease,   and   so   on.   It   is   plain   on   this hypothesis   that   a   race   afflicted   by   any   disease   should   undergo evolution   or   degeneration,   evolution   if   the   disease   tends   to strengthen the individual against subsequent attacks by conferring immunity, degeneration if it tends to weaken him.   On the other hand, if the effects are not transmissible, then a race afflicted by deadly   disease   would   change   equally,   would   undergo   evolution equally, but not degeneration.   For men differ individually in their power of resisting disease.   Deadly disease is therefore a selective agency.   It weeds out the less resistant to it, leaving the race to the more resistant.   Therefore, on this other, this Darwinian hypothesis, a race afflicted by a disease should grow more and more resistant to it.   Moreover, since resisting power against one disease does not imply resisting power against any other—for example, since resisting power against consumption does not imply resisting power against malaria—every   race   should   grow   resistant   only   against   the   particular death-dealing diseases by which it is afflicted."[2]

If I have made myself clear, it will be seen that the evolution of immunity to any given noxious agency is brought about by precisely similar means as the evolution of any other human characteristic.

The naturally immune are selected to carry on the race, in so far

1 "Alcoholism."—G. Archdall Reid, pp. 9, 51.   2 Ibid, pp. 34, 35.

as that special agency is a selecting one. Those who can acquire immunity likewise survive, and that power is reproduced in their offspring. In the one case an inborn immunity is evolved, in the other an increased capacity of resistance. In both cases the result of continuous selection by disease, acting on any race, is to make that race resistant just in proportion to its past experience of the condition.

To tubercle then we are becoming naturally immune; the susceptible have perished and are perishing. To measles we are still susceptible, but its dangers are a thing of the past, and the longer we are subjected to the infection the more resistant shall we become, because the least resistant perish.

And, so, taking resistance to disease as the test of fitness, the evolution of immunity is the result of the operation in the sphere of disease, as in all other spheres, of the great law of natural selection by survival of the fittest.

GLANDERS.[1]

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HISTORY.

ACCORDING to Bass,[2] the earliest recorded reference to glanders is found in the writings of Aristotle, who describes the only disease of the ass under the term μηλις (melis). The description is meagre, since the passage merely states that the disease begins about the head, with a purulent discharge from the nose, and proves fatal when it extends to the lungs. But the same author has also been credited with a knowledge of farcy owing to his statement that horses develop abscesses.

In the writings of the next six centuries no reference to either glanders or farcy has been identified, and the disease is next mentioned by Apsyrtus, who lived during the fourth century of the Christian era. Apsyrtus appears to have employed the term μαλις (malis) in somewhat the same sense as the English word distemper, and he distinguished four forms of the disease, viz., moist, dry, articular, and subcutaneous. It is probable that the first of these was glanders, and the second farcy. Assuming this to be correct, Apsyrtus, as Bass points out, was not only acquainted with glanders but also believed that the disease was contagious, and actually recommended the segregation of affected animals in order to prevent the disease from spreading. It is worth noting, however, that, like many subsequent writers up to comparatively recent times, Apsyrtus described glanders as an easily curable disease.

Glanders appears also to have been known to Hippocrates, who was a contemporary of Apsyrtus, but his writings contain no reference to the disease except a prescription for it. He is also said to have pronounced the disease incurable during its advanced stages.

Vegetius, who wrote a century later than Apsyrtus, used the term *malleus* in a very loose or comprehensive sense. He distinguished seven different varieties of the disease so-named, and two of these, viz., malleus humidus and malleus farciminosus, have respectively been identified with our glanders and farcy. His account of the symptoms of these affections is sufficiently full to make it certain that he was well acquainted with both of them. He considered farcy easily curable, but described a bloody character or a saffron-yellow colour of the nasal discharge in glanders as an indication that the disease was likely to prove fatal. Much more interesting is the fact that Vegetius recognised that glanders was a contagious disease, and indeed in this respect he went considerably further than Apsyrtus, since he recommended the isolation of the suspected as well as of the actually diseased. So convinced was he of the danger of contagion that he advised the burial of glandered carcases in places to which animals should not have access. In this Vegetius no doubt went beyond the necessities of the case, but in his estimate of the risks of contagion from living diseased animals he appears to have been more than a thousand years in advance of his time. As an offset to this, however, it ought to be mentioned that he regarded bad air as the great originator of the disease. The air which was supposed to exert this deleterious influence was not the atmosphere rendered foul by over-crowding or animal exhalations, for the west and south winds were supposed by him to be generally dangerous alike to animals and man.

Glanders and farcy are mentioned in the writings of various authors who lived during the following ten centuries, but none of the accounts of the disease given during this period deserve to be quoted.

Fayser and Seuter, both of whom lived during the sixteenth century, recognised the contagious character of glanders, and the former was aware that farcy was transmissible from diseased to healthy horses, while the latter appears to have been the first to place on record the view, not yet universally abandoned, that the contagion may hang about an infected stable for a period of many months.

An English contemporary of the two authors last mentioned, named Fitzherbert, wrote a *Boke on Husbandry* in 1523, and this contains the first reference to glanders and farcy which has yet been unearthed from English literature.[1] It is interesting on this account, and also because it proves that the contagious character of farcy was then recognised in England. The passages relating to glanders and farcy are as follows :—

"Glaunders is a desease that may be mended and cometh of a heate, and a sodeyne cold, and appereth at his nosethrylles, and between his chall (jowl or jaw) bones. Mournynge on the chyne is a desease incurable, and it appereth at his nosethryll lyke oke water. A glaunder, when it breaketh, is lyke matter. Broken-winded and pursifness is but shorte blowynge . . . The Farcyon is but an yll soraunce, and maie well be cured in the begynnynge, and wyll appere in dyverse places of his bodye, and there wyll ryse pymples as much as halfe a walnutshell, and they will followe a veyne, and wyll breake by it selfe ; and as many horses as do playe with him that is sore,

[1] My attention was called to this by Sir Ernest Clarke, Secretary of the Royal Agricultural Society of England.

and gnappe of the matter that runneth out of the sore, shall haue the same sorance within a moneth after : and therefore kepe the sycke from the whole. And if that sorance be not cured betyme, he wyll dye of it."

In *Markham's Masterpiece*, which is a work on farriery published in 1662, both glanders and farcy are dealt with at considerable length. The former is described as a "running impostume ingendred either by cold or by famine, or by long thirst, or by eating corrupt and musty meat." From the point of view of prognosis, Markham attaches great importance to the colour of the nasal discharge, and it is interesting to find him agreeing with Vegetius that a yellow tint is of bad omen. He says : "If it be yellow, separate him from the sound horses, for he is infectious yet to be cured. If that yellow be mixed with blood, it is with much difficulty helped, or if the matter be like unto saffron, the horse is hardly to be saved." Farcy is dealt with apart from glanders, and it is not suggested that there is any connection between them. A most heterogeneous list of causes, including the bites of dogs and contact of swine with the horse's legs, are enumerated. The infectious character of the sores was, however, recognised, and it is said that "as many horses as do gnaw or gnapp upon the horse infected will within one moneth have the same disease, or if the horse infected do bite another, he will infect him also." Markham therefore advises that "any horse troubled with this sorrance" should be isolated.

Solleysel, who was a member of the Royal Academy of Paris, and equerry to the King of France, published in 1667 a large work on horsemanship and farriery entitled *Le Parfait Mareschal*, and the eighth edition of this was translated into English by Sir William Hope, Deputy-Lieutenant of the Castle of Edinburgh. A copy of the second edition of the English version, published in 1717, is in the library of the Royal Veterinary College, and the following passages are quoted from it :—

"The immediate cause of the glanders is frequently an ulcer in the lungs (rarely in the kidneys), from which subtle and malignant vapours are sent up to the brain. . . . This is the most contagious distemper to which horses are obnoxious, for not only it communicates its venom at a small distance, but infects the very air, and seizes on all the horses that are under the same roof with him that languishes under it. And therefore as soon as you perceive the least sign of the glanders, you must separate the sick horse from all his companions, and not suffer him to drink out of the same pail with them, especially when the disease is malignant ; for there are several kinds of glanders, some of which are not so extremely infectious as others, but there are none of them that ought not to be suspected.

"I'm persuaded that this distemper proceeds from a cold cause, and I do not at all doubt of the extreme difficulty of the cure. All its various kinds are only distinguished by a greater or less degree of malignity ; and it will be found that all those who pretend to have cured the glanders, have only cured either the strangles, cold, or some less malignant sort of glanders ; for certainly these cures are very rare and (perhaps I might justly say) impossible."

Solleysel treats of farcy apart from glanders, and it is curious to note that, although he did not suggest any connection between the

two conditions, he was aware that farcy was apt to be accompanied
by a running at the nose.   He says: "The most dangerous and
stubborn kind of farcin is that which is accompanied with a running
at the nose, for as soon as you perceive that symptom you may con-
clude that your horse's death approaches, especially if his appetite
be lost, and the matter that runs out be mixed with blood.   The
number of those which escape is so small that all horses in this con-
dition may be given over for desperate, unless the running at the
nose proceed from the strangles, and even in that case the disease is
usually fatal."

In 1751 William Gibson, a surgeon of Duke Street, Grosvenor
Square, published *A New Treatise on the Diseases of Horses*, in which,
as in Markham's work, glanders and farcy are treated as two distinct
diseases.   The "most common and usual kind" of glanders is vaguely
said to come from a bad disposition of the blood, but a glanders
arising from infection is recognised, and is said to be more amenable
to treatment than when it is the effect of a long-continued ill habit.
Farcy is described as a disease of the veins, and its common causes
are said to be errors of diet—drinking cold water when heated, cool-
ing suddenly after exercise, etc.   It is, however, added that "some-
times the farcy may proceed from infection when it happens to be
a sickly time among horses, or when it is epidemical ; however, this
is but seldom, for the farcy in the commoner way is rarely infectious,
or any ways hurtful to other horses, as we see by daily experience."

During the latter half of the eighteenth century many works on
farriery were published by French, German, and other Continental
authors ; but the views of most of them on the subject of glanders
and farcy show no real advance with respect to a knowledge of the
cause of the disease.   By this time the fact that glanders might be
spread by contagion appears to have become generally recognised,
but it was universally believed that it might have other causes, and
glanders and farcy were not recognised as being identical, except
that both might be engendered by the same hygienic or dietetic
errors.

Erik Viborg, a Dane, who published a work on veterinary surgery
and agriculture towards the close of the eighteenth century, appears
to have been the first to contend that glanders and farcy had a
common contagium, and to prove by experiment that glanderous
discharge might be employed to infect a horse with farcy by cutane-
ous inoculation, and that farcy so set up might develop into glanders.
He had also determined by experiment that glanderous materials
cease to be infective when dessicated.   He attached great importance
to disinfection of contaminated stables, and was alive to the fact that
the disease might be spread by horses that appeared to have been
cured of an attack of farcy.

Saint Bel, who was Principal of the Royal Veterinary College for
two years after its foundation in 1791, was well acquainted with the
contagious nature of glanders, and carried out experiments intended
to ascertain the methods of its transmission.   In his posthumous
Essay on the Glanders, published in 1797, he states that "the infection
may be received by one animal rubbing against another, by the
breath, and by a sound horse eating the slavered food of a diseased
one ; but not by water, unless the virus is swallowed with it ; neither

by inoculating the body with the morbific matter." The identity of glanders and farcy does not appear to have occurred to him.

Coleman, who succeeded Saint Bel in 1793 and died in 1839, regarded some " atmospherical impurity " as the great cause of glanders, but he had himself produced experimental evidence of the identity of glanders and farcy by inducing farcy through inoculation with the poison of acute glanders, and *vice versa.* From the beginning of last century to well past its middle, discussion with regard to the etiology of glanders turned not so much on the question whether the disease could be spread by contagion as on the question whether it had other modes of origin, and whether contagion was the common cause. Coleman did not believe that one horse in ten thousand caught the disease from contagion. He thought that imperfect ventilation and other unhygienic conditions in stables could generate glanders, and that, in reality, this was the cause at work even where the disease assumed epizootic characters. In France and Germany, at the same period, the predominant opinions with regard to the cause of glanders were in agreement with those of Coleman. After Coleman's death opinions in this country gradually changed with regard to the relative importance of spontaneous generation and contagion in the causation of glanders and farcy, but the view that the disease might develop amid insanitary surroundings and independently of contagion has only died out within the last twenty years.

The issue of the controversy with regard to the possibility of " spontaneous generation " of living things, and the discovery of the causal organism of the disease by Löffler and Schütz in 1882, no doubt contributed largely to this change of opinion, but the gradual abandonment of the view that glanders might originate independently of contagion has certainly been in great measure due to other circumstances. In this and other European countries the effect of repressive measures based on the belief that glanders, whether sometimes originated spontaneously or not, always tends to spread by contagion, gradually gave the country, as contrasted with the towns, a comparative freedom from glanders, and owing to the altered incidence of the disease it gradually became possible to observe that in the great majority of cases outbreaks in non-infected localities were traceable to the introduction of diseased horses, or at least to horses from infected places. In this connection it must be remembered that, although belief in the possibility of spontaneous generation of living things has been made untenable, it is still permissible to believe that a disease which is ordinarily contagious may sometimes originate sporadically. That, however, is a point to which I shall refer again immediately in dealing with the biology of the glanders bacillus. Meanwhile I only wish to point out, that if there is now general agreement that glanders has no other cause than contagion, that is mainly because the distribution of the disease throughout the world has enabled us to trace the operation of contagion in a way that was impossible when the disease was more widely disseminated.

BACILLUS MALLEI.

Habit.—The causal microbe of glanders and farcy, which was first accurately described by Löffler and Schütz, must be considered an obligatory parasite, with the equine species for what may be called

its normal host. Löffler found that no growth took place in artificial cultures at a temperature of 68° F., and that it was slow and meagre at 72° F. While it is thus possible that in summer the bacillus may occasionally propagate in the outer world, it is certain that throughout the greater part of the year in temperate climates it is denied the necessary temperature even in the stable. On the other hand, although the optimum temperature is that of the mammalian body, growth is fairly rapid at all temperatures between 90° and 100° F., and it has therefore been suggested that in tropical countries the glanders bacillus may sometimes be able to maintain a saprophytic existence in the soil or in the excreta of horses in infected stables. As a matter of fact, however, nothing in connection with the geographical distribution of glanders lends any support to this view. Although the prevailing external temperature may be considerably above the minimum necessary for the growth of the glanders bacillus, that organism must always in the outer world find itself in competition with hardier and more rapidly growing saprophytic bacteria, by which it is certain to be soon crowded out of existence. Glanders is not specially a disease of countries with a high temperature, and, as a matter of fact, London is probably the place in which the disease is at the present time most prevalent. Here, it is true, the largest number of cases are annually reported between June and September, but the excess above the average of the colder months is not great, and Hunting is probably correct in ascribing the annual summer increase to the more exhausting character of the work to which city horses are subjected during the hottest period of the year.

The habit of life of the glanders bacillus may be summed up by saying that at the present day its existence is bound up with that of the equine species. If the horse became an extinct animal the glanders bacillus would perish with it. The occasional cases of glanders which occur in man and other species than the horse are all traceable to infection from equines, and, broadly speaking, such cases play no part in maintaining the existence of the glanders bacillus.

Distribution in the body.—The glanders bacillus is a tissue parasite, and in all cases of glanders and farcy in the horse it is almost entirely confined to the lesions and the discharges from them. It is no doubt transported by the blood as well as by the lymph, but the disease in the horse is never septicæmic. Even in acute cases of glanders the bacilli are so sparingly present in the blood that their discovery by microscopic or cultural methods is nearly always impossible, and even considerable quantities of blood usually fail to transmit the disease by inoculation. The bacilli are most abundant in recent acute lesions, but even in these they are usually not enormously abundant, as compared with the richness of some other bacterial lesions in their causal organisms. Smears from farcy pus or glanders nodules of the horse never show immense numbers of the bacilli, and the small number of colonies which are frequently obtained from potato surfaces inoculated with a good loopful of farcy pus is in agreement with the apparent scarcity of the bacilli in film preparations.

Morphology.—The glanders bacillus, as it is found in natural lesions in the horse and other animals, is a cylindrical rod, varying between 2 and 5 microns in length and about half a micron in breadth. The ends of the bacilli are rounded, and the majority of the rods are

straight, though some are slightly curved. When stained with aqueous solution of methylene-blue some of the rods take on the colour uniformly, but the blue tint is not so deep as it is in many other bacteria when similarly stained. Some of the rods exhibit the bi-polar method of staining, and others show minute alternating stained and unstained segments, the appearance of such rods being somewhat similar to the "beading" of tubercle bacilli. This appearance may be indicative of degeneration, and, in view of the ease with which the bacilli are killed by heat and disinfectants, the unstained particles cannot be regarded as spores. In the lesions of glanders and farcy the bacilli are usually irregularly scattered, and exhibit no marked tendency to form colonies, or to group themselves in any particular way. That at least is true of the lesions in the horse, but Mayer found in the lesions of the guinea-pig aggregations of bacilli recalling the colonies of actinomyces. The lesions, however, in which these *bizarre* forms were met with had been set up by intraperitoneal inoculation of bacilli suspended in a relatively large quantity of melted butter.

In artificial cultures the early growth is almost always composed of short rods, which appears to be a little thicker than those found in lesions. This type of growth is usually maintained in old potato cultures, but the surface growth of old bouillon cultures is often largely composed of thread-like forms. Many of these are of great length, stretching across the whole field of the microscope. These filaments do not exhibit any regular segmentation, but some show distinct lateral twigs, and occasionally the ends of such threads show a club-like swelling. In very old cultures in bouillon these filaments often stain faintly, but show in their substance minute more deeply stained particles. This probably denotes death or degeneration of the filaments. Levy, Conradi, Galli-Valerio, and others have called attention to this tendency of the glanders bacillus to assume the form of threads which occasionally exhibit a true branching and terminate in club-like swellings, and have from these morphological characters inferred its relationship with the actinomyces group of organisms.

Cultural Characters.—The glanders bacillus is an organism which lends itself well to artificial cultivation, and, when exposed to the air and kept at temperatures approachiug that of the body, it grows well in or on nearly all the culture media in common use. The greatest interest attaches to its growth on potato, as on that medium it exhibits characters which are practically diagnostic. When the surface of a sterilised potato slice is rubbed over with a loopful of farcy pus or material from the centre of a glanders nodule, and the tube is incubated at 97-100° F., the resulting growth is usually not distinctly visible till the third day. On that day or the following one the inoculated surface will show a continuous growth or one composed of small raised discrete colonies. In either case the growth is remarkably honey-like in respect of colour (amber-yellow), translucency, and viscidity when tested by touching with the platinum needle. With continued incubation the growth increases in thickness, becomes opaque, and deepens in colour, so that after a week or ten days it has assumed a fawn colour. Still later it becomes reddish-brown, and finally it assumes a deep chocolate-brown tint. Not every tube gives

this last colour, especially if the incubation is continued at the body temperature. As a further character of the growth on potato, it ought to be added that it is odourless, and remains strictly limited to the surface on which the seed material was implanted. There is no diffusion of colour into the surrounding potato substance. The diagnostic importance of these characters may be expressed by saying that any organism which exhibits them, and which on microscopic examination is found to have the morphological characters previously described, may confidently be pronounced to be the glanders bacillus. Future investigations may show that there exist in nature other bacteria which cannot by these characters be distinguished from the glanders bacillus, but none likely to be thus confounded with the bacillus mallei has yet been encountered.

In bouillon, and still better in glycerine-bouillon (5 per cent.), the glanders bacillus grows copiously, but with appearances that are much less characteristic. Bouillon cultures within four or five days become distinctly and uniformly turbid. This is true even when a litre flask of the medium is inoculated. After a week or more a whitish scum begins to form on the surface of the medium, and after three weeks the surface of the bouillon in flasks is generally largely covered by this surface growth. The growth may be almost continuous, but at an earlier stage it is composed of circular floating patches, and ultimately the growth has a faint yellowish tinge. When an attempt is made to lift the surface growth on the loop of a platinum needle it is found to have a slimy consistence, and it is easily broken up by shaking. Along with this surface scum a quantity of growth collects at the bottom of the liquid. When the liquid is agitated this appears somewhat ropy, but, like the surface growth, it is easily broken up. As previously observed, these appearances are less characteristic than those exhibited by the growth on potato, but, nevertheless, the sum of them is fairly distinctive of the glanders bacillus.

The glanders bacillus also grows on the surface of agar and solid blood serum, the growth being at first white and ultimately yellow or brownish.

Staining Reactions. — All bacteriologists admit the difficulty of staining the glanders bacillus when it is included in the tissues, but there is no real difficulty in staining cover-glass films of glanderous or farcy material so as to reveal the presence of the bacilli. The stain which was originally recommended for the purpose by Löffler was a faintly alkaline aqueo-alcoholic solution of methylene-blue, since generally known as Löffler's alkaline methylene-blue. This stain, according to Löffler's prescription, is made by adding 30 cc. of a concentrated alcoholic solution of methylene-blue to 100 cc. of a dilute aqueous solution of caustic potash (1 of the alkali in 10,000 of water). Sections are immersed in the stain for five minutes, then treated for a few seconds with 1 per cent. solution of acetic acid in water, dehydrated in alcohol, and clarified in cedar oil. Löffler's solution is, I believe, still widely used for the demonstration of glanders bacilli, but it has no special advantage for cover-glass films, and it leaves much to be desired for the demonstration of the bacilli in sections. In ordinary thin smears of farcy pus or material from glanders nodules on cover glasses, glanders bacilli, like most other

bacteria, stain instantly in simple aqueous solutions of any of the basic aniline dyes in common use (methylene-blue, gentian violet, fuchsin) and the only difficulty in recognising them lies in the presence of nuclear detritus which masks them or may be mistaken for them. This difficulty is not entirely overcome by subsequent treatment with 1 per cent. acetic acid, which has only a weak decolourising effect on the nuclei and granular detritus present in the film, but, contrary to the general impression, much stronger solutions of this acid may be employed without any risk of completely decolourising glanders bacilli. With the object of making the bacilli more distinct I was induced to use acid solutions of increasing strength, and was surprised to find that, in cover-glass films, glanders bacilli stained with methylene-blue remained fairly well stained when treated for several seconds with a 10 per cent solution of acetic acid in water. There is, however, no occasion to use such a strong solution, for a 4 or 5 per cent. solution suffices to decolourise the film to such a degree as to render any bacilli present remarkably plain. Whether the films have been fixed by heat or by alcohol, the bacilli remain well stained when thus treated.

There is no reason to suppose that glanders bacilli embedded in the tissues are less retentive of the stain than those contained in cover-glass films, but the difficulty in the case of sections arises from (1) the fact that throughout the greater part of every glanders or farcy lesion the bacilli lie amid an abundance of nuclear detritus (resulting from the chromatexis referred to later on), and (2) from the fact that the alcohol employed in dehydrating sections preparatory to mounting them in balsam exerts a rather powerful decolourising effect on stained glanders bacilli. On this account it is difficult to obtain satisfactory preparations of sections by the method of Löffler. The bacilli are sometimes fairly distinct at the margins of the lesions, but elsewhere they are apt to be quite indistinguishable amid the heap of chromatin threads and particles.

The use of a solution of tannin after the sections have been washed in water or weak acetic acid solutions, as in the methods of Nicolle and Unna, greatly improves the result by fixing the methylene-blue in the bacilli, and thus diminishing the decolourising action of the alcohol which has subsequently to be used for dehydration ; but, if only 1 per cent. acetic acid is employed for the preliminary washing of the stained sections, the nuclear detritus is apt to retain so much of the blue stain as to make it difficult or impossible to distinguish the bacilli in many parts of the lesions. After a trial of many different methods, I have obtained the best results by the following procedure :—

The thinnest possible sections of lesions hardened in alcohol are made after embedding in paraffin, and fixed on glass slides. For this purpose no fixative is required, the sections adhering perfectly when floated on to the slide in water a few degrees below the melting point of the paraffin, and afterwards dried for a few hours in an incubator at 37° C. After removal of the paraffin in the customary manner (xylol followed by alcohol), the sections are stained for half an hour with a solution of methylene-blue. This may be either Kühne's carbolised solution, Löffler's alkaline solution, or simply 1 per cent. solution in water 90 parts and alcohol 10 parts. I have not been able

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to satisfy myself that the last of these is in any way inferior to the
other two. The sections are washed in water to remove the super-
fluous stain, and they are then treated for a few seconds with 4 per
cent. solution of acetic acid in water, followed by plain water. The
acid solution almost entirely decolourises any normal tissue surround-
ing the lesion, and the latter is reduced to a pale blue. The section
is now covered with a saturated solution of tannic acid in water for
fifteen minutes, after which it is well washed in water, and flooded for
fifteen to thirty seconds with a 1 per cent. solution of acid fuchsin in
water. The subsequent procedure includes washing in water, dehy-
dration in alcohol, clarifying in cedar oil, and mounting in xylol
balsam.

In sections thus treated the nuclei and nuclear detritus in the lesion
have almost entirely lost the blue stain, and the tissues are for the
most part of a faint red, while the bacilli are of a light blue colour.

In concluding the subject of staining it ought to be said that no
method for staining glanders bacilli in the tissues ought to be tested
except on sections which are known to contain the bacilli in consider-
able numbers, and this assurance is best obtained by examining
smear preparations from the nodules or other lesions prior to
hardening in alcohol. In many cases, both in the horse and in the
guinea-pig, the stainable bacilli are very sparing in number in the
older lesions, just as is frequently the case in old tuberculous
lesions.

Resistance to germicides.—An opinion which has long been very
generally held is that the efficient disinfection of glanders stables is a
matter of very considerable difficulty, but this view is not in accord
with the results of experiments bearing on the vitality of the glanders
bacillus outside the body. In flasks of bouillon incubated at the body
temperature growth comes to an end in three or four weeks, and if the
incubation is continued at that temperature the culture is frequently
dead in another month. Even potato cultures started at 37° C. and then
left at the room temperature often prove to be dead when one tries to
start fresh cultures from them after a month or two. Young bouillon
cultures are soon killed by exposure to bright sunlight, and Löffler
found that an exposure of ten minutes at a temperature of 55° C. was
fatal to artificially cultivated glanders bacilli suspended in distilled
water. The same author also proved that glanders bacilli are com-
paratively sensitive to chemical disinfectants. In silk thread experi-
ments a 3 per cent. solution of carbolic acid was found to be fatal in
five minutes. The same result was obtained in two minutes with a
1 per cent. solution of permanganate of potash, and with a 1 in 5000
solution of corrosive sublimate. Complete desiccation of nasal dis-
charge, farcy pus, or bacilli from artificial cultures, at the body
temperature, is frequently fatal in twenty-four to forty-eight hours ;
but, as bearing on the question of aerial infection, it is deserving of
notice that Löffler found that silk threads impregnated with bacilli
in the condensation water of a serum culture when dried for a day in
the incubator (and then apparently kept at the room temperature)
proved infective to field mice as long as eighty days afterwards.

As previously remarked, the general result of bacteriological ex-
periments is not in keeping with the view that the disinfection of
contaminated stables is hard to accomplish, and at first sight it

appears difficult to account for what was until lately a popular be-
lief—viz., that nothing short of the destruction and rebuilding of an
infected stable was certain to prevent the recurrence of outbreaks.
However, in the light of knowledge acquired within recent years it
is easy to see how the facts in this connection come to be misinter-
preted.   Formerly an outbreak was generally supposed to be at an
end when all the apparently affected horses had been killed or
removed, and when the disease recurred a few months afterwards
it was natural to conclude that the measures of cleansing and dis-
infection had failed to get rid of the virus in the floor, walls, or fittings
of the stable.   The inefficiency of disinfection seemed to be still more
clearly demonstrated when the disease recurred after re-stocking the
stable with apparently healthy horses.   It need not be affirmed that
there is no risk from perfunctory disinfection, but there can be little
doubt that in most of the cases relied upon to prove the difficulty of
destroying the virus in the stable, the recurrence of the disease was
due to infected but apparently healthy horses having been left over
from the previous outbreak or introduced when the stable was re-
stocked.

METHOD OF INFECTION.

It is beyond dispute that glanders and farcy are readily trans-
missible by ingestion and inoculation.   That has been established
by numerous experiments, both old and recent.   It is also well
established that intra-uterine infection of the foal is possible when
the mare is glandered.   There is, however, a wide difference of
opinion with regard to the common method of natural, as opposed
to experimental, transmission, and especially with regard to the
question whether in the majority of cases horses are infected by the
inhalation of bacilli suspended in the stable air or by the ingestion
of bacilli with their food or water.   Among veterinary surgeons in
this country the prevalent opinion has, I believe, always been in
favour of what may be called the ingestion theory of infection, and
the evidence relied upon is partly clinical, partly experimental, and
partly drawn from what is known with regard to the weak resistance
which the glanders virus offers to dessication.   On the other hand,
the supporters of the inhalation theory cannot, so far as I know, cite
any direct experimental evidence ; but they rely mainly on the dis-
tribution of the lesions in natural cases of glanders, holding that this
is inconsistent with infection by ingestion, and in keeping with the
view that the virus generally enters the horse's body by way of the
air passages.

The clinical evidence in favour of the ingestion theory must be
adjudged inconclusive.   It is said that observation of the way in
which the disease spreads in an infected stud points to nose-bags,
mangers, and watering pails or troughs as the principal or exclusive
vehicles by which the contagion is carried, and it is also stated by
some that, since in London the disease occasionally breaks out in
healthy studs where every possible source of infection except public
drinking troughs has been excluded, the latter must be held respon-
sible for the mischief, and even accorded an important rôle in the
dissemination of the disease.   I say this evidence must be considered
inconclusive, because, with the exception of the drinking troughs, the

vehicles which are alleged to spread the disease by ingestion are practically never in operation in circumstances that exclude a distinct possibility of infection by inhalation. That point is so obvious that it only needs to be mentioned. As regards public drinking troughs also, it may be affirmed that no one has yet recorded an outbreak where the circumstances compelled one to admit that the bare possibility of infection in any other way had been excluded.

The experimental evidence in favour of infection by ingestion cannot be dismissed so easily. The late M. Nocard was a strong believer in this view of natural infection, and he held that certain experiments which were carried out at his instigation in 1896 yielded results that were in this connection conclusive. In these experiments four horses which did not react to mallein, and which belonged to a regiment in which glanders had not been observed for ten years, were given the whole of a potato culture of virulent glanders bacilli mixed with half a pail of water. Fifteen days afterwards two of these horses were killed, and in both cases the *post-mortem* examination showed that the lung tissue was crammed with miliary nodules in all stages of development. The two remaining horses were killed at the end of three months, after they had for some time presented all the clinical signs of chronic glanders, and in them also the lungs were found to be crammed with nodules in all stages of evolution. Nocard concluded that many of these pulmonary nodules were the result of auto-infection, owing to the horses having daily swallowed glanders bacilli from their own discharges.

In a second series of experiments twelve horses were made to ingest the growth of three potato cultures of glanders bacilli mixed with water. All of them reacted to mallein fifteen days afterwards, and five of them, killed at different intervals after infection, were found to be glandered.

Nocard, in maintaining that these experiments proved that ingestion is the common method of infection in natural cases of glanders, appears to have misunderstood the position of those who oppose this view. The experiments merely proved that it is possible to infect horses with glanders by ingestion, and that in horses so infected glanderous lesions develop in the lungs, and sometimes also in the Schneiderean mucous membrane and submaxillary lymphatic glands ; but that had never been denied. Strange to say, Nocard's account of the experiments was silent as to the presence or absence of glanderous lesions in connection with the abdominal organs. On account of this omission, the experiments left the question as undecided as before. It was as if an experimenter who wished to disprove the possibility of tuberculous infection by inhalation were to feed animals with tuberculous materials and maintain that he had proved his point by demonstrating the presence of tuberculous lesions in the lungs when the animals were killed.

Professor Schütz, of the Berlin Veterinary College, in the following year repeated Nocard's experiments, but varied them by administering the infective material in the form of a bolus coated with gelatine. Assuming that the absence of any mention of abdominal lesions in Nocard's account of the French experiments was not an omission, but due to the fact that no such lesions were present, the experiments carried out by Schütz had very different results. In the first of these

a horse was given in bolus the whole of the glanders bacilli furnished by twenty potato and twenty-two glycerine-agar cultures. The horse was killed fourteen days afterwards, and the *post-mortem* examination disclosed more or less conspicuous glanderous lesions in the mesenteric and colic lymphatic glands, liver, spleen, lungs, and intestines (one pea-sized nodule in a Peyer's patch, and two of the same size in the colon). In a second experiment the infective material administered in the same way was furnished by sixteen potato and twenty-four glycerine-agar cultures. The horse was killed thirteen days afterwards, and again, in addition to pulmonary nodules, marked lesions were present in the abdominal cavity, including pea-sized nodules in the intestine, enlargement of the mesenteric glands (with pin-head areas of degeneration), and nodules in the spleen and liver.

In a third experiment a mare was made to ingest the product of sixteen potato and twenty-four glycerine-agar cultures of glanders bacilli, and in this case the bolus broke in the mouth. Thirteen days afterwards the mare was killed, and again at the *post-mortem* examination glanderous lesions were found in the bowel, mesenteric glands and liver, as well as in the lungs. Ulcers were also present in the pharynx, and nodules in the submaxillary and pharyngeal lymphatic glands.

In a fourth experiment a horse was given in bolus the tenth part of a loopful of a glycerine-agar culture, but no glanderous lesions were found in this animal when it was killed twenty-five days afterwards.

The last experiment was similar to the preceding one, save that the small dose of bacilli was given daily from the 13th to the 17th August. The horse was killed on the 8th October following, and at the *post-mortem* examination conspicuous glanders lesions were found in the abdominal lymphatic glands, liver, and spleen, as well as in the lungs.

It cannot be held that these experiments lent any support to the view that ingestion is the common or exclusive method of infection, for it is quite certain that in the majority of glandered horses killed before the development of outward symptoms, no lesions are discoverable with the naked eye in the spleen, liver, or abdominal lymphatic glands. In a considerable proportion of cases of advanced glanders lesions are present there, especially in the spleen and liver, but rarely in the mesenteric or colic glands. As regards the distribution of the lesions, these experimental cases are so far out of the common as to make it impossible to use them in support of the ingestion theory of natural infection. However, it must be admitted that the conditions of the first three experiments bore no resemblance to anything that can ever happen in natural circumstances, as the dose of infective material employed was enormous. The last experiment is not open to that objection, and had it been often repeated with like results it would have been difficult to maintain any longer that ingestion is the common method of infection.

I have myself infected four horses with glanders by causing them to swallow glanders bacilli. In each case the quantity of infective material administered was approximately the same, viz., the whole of a potato culture which was the second generation from a natural case of glanders in a horse, and which had been incubated at $37°$ C. for

five days. A cylindrical hole was bored through a piece of very tough carrot, and into this the glanders growth scraped from the surface of the slice of potato was introduced. Each end of the cylindrical hole was then closed with a very tight-fitting cork. Care was taken to avoid touching the outside of the piece of carrot in introducing the culture, but, as an additional precaution, the ends of the piece were mopped with a strong solution of corrosive sublimate. The bolus thus made up was administered by hand by a member of my post-graduate course, Mr Byrne, M.R.C.V.S., and in each case it was swallowed without crushing in the mouth. All the four experimental animals were aged ponies in rather poor condition.

CASE I.—Was submitted to a preliminary test with mallein, and it did not react. It had to be killed on the eighth day after infection, as it was prostrate and unable to stand. The *post-mortem* examination, which was begun immediately after death, disclosed the following lesions :—

The whole of the mesenteric glands were congested and considerably enlarged, though none was as large as a pigeon's egg. Tubercle-like opaque points were visible in some of them. The lymphatic glands belonging to the double colon were also enlarged, the largest being about the size of a garden bean, and small, opaque, necrotic-looking areas were present in many of them. The spleen was normal in size. One nodule, about the size of a barley grain, and presenting the usual appearance of a glanders nodule in that organ, was present under its capsule. The kidneys were normal, but some old, white, calcified nodules were present under the capsule of the liver.

The mucous membrane lining the right sac of the stomach was generally congested, and beset with raised areas about one-fourth to one-third of an inch in diameter. The surface of these was above the level of the rest of the mucous membrane, and appeared to have suffered a slight loss of substance.

In the small intestine, under the mucous membrane, there were two slightly projecting nodules, the larger about the size of a vetch pea. One nodule about the size of a pea was present under the mucous membrane of the cæcum. The contents of these nodules were thick and rather dry, like necrotic material or inspissated pus.

The lungs contained some hundreds of nodules with the ordinary appearance of glanders nodules. They varied in size between a filbert nut and a barley grain. On section the nodules showed a central grey or dirty-white opaque area and a peripheral congested or hæmorrhagic zone. No lesions were visible in the thoracic lymphatic glands.

Numerous small nodules were present under the mucous membrane of the nose, both on the septum nasi and the turbinated bones. The tongue, fauces, pharynx, larynx, and trachea were normal, as were also the submaxillary and pharyngeal lymphatic glands. All the body lymphatic glands were examined, and all appeared to be normal, except the left precrural group. This was a little enlarged, and showed one or two opaque, necrotic-looking areas.

Glanders bacilli were cultivated on potato from a lung nodule, a mesenteric gland, the left precrural gland, and one of the submucous nodules in the small bowel (only one thus tested). Two tubes inoculated from the submucous nodule in the cæcum yielded no growth.

The splenic nodule was not used for cultures, but was reserved for microscopic examination. It was found to have the histology of a glanders nodule, and showed pronounced chromatotexis.

CASE II.—This pony when tested prior to experiment did not react. It was again tested on the thirteenth day after infection, and it then had a rise of temperature and a local reaction indicating that it was the subject of glanders. It was killed on the fifteenth day of the experiment, and the *post-mortem* examination showed the following :—

Nothing abnormal was observable in any of the abdominal lymphatic glands, except that the mesenteric group was congested (muscle-coloured). In the mesentery, below the mesenteric glands, there was a nodule a little larger than a garden bean. The stomach and intestines, both large and small, showed no abnormality, although they were carefully searched both as regards their exterior and their interior. The spleen was normal in size, but on its outer side, 2 inches from the base, a pea-sized nodule, resembling a glanders nodule, was present under the capsule. The liver was normal save for some thready fibrous growths on its surface, and the presence of a number of pin-head white fibrous-looking nodules under its capsule. The kidneys and bladder were normal. The lungs contained five nodules with the characters of glanders nodules, the largest being about the size of a vetch pea. No lesions were present in the thoracic lymphatic glands. No other lesions were discovered anywhere, although all the parts mentioned in the account of the preceding case were examined. Glanders bacilli were cultivated on potato from one of the lung nodules (the only one tested), and from the splenic nodule. The above-mentioned nodule found in the mesentery was found to have a thick fibrous-capsule, and central thick, greyish contents, which, when the nodule was punctured, could be squeezed out in the form of worm. Potato tubes inoculated with this material yielded a copious growth, which was shining white with a faint trace of yellow, and composed of small bacilli, probably the bacillus coli.

CASE III. did not react when tested prior to the experiment, but when tested twelve days after infection the temperature rose 3·7° F., and there was a considerable local reaction. It was killed on the twentieth day after it received the bolus containing glanders bacilli, and the *post-mortem* examination revealed the following :—

The whole of the mesenteric glands were quite normal in size, free from congestion, and showed no nodules or signs of necrosis. The glands of the double colon appeared normal in size, with the possible exception of the one lying nearest to the mesenteric group. This was about the size of a garden bean, and its substance appeared normal. On the course of the colic vessels, on the second part of the double colon, there were two nodules, about an inch and a half apart. The larger of those was about the size of a filbert nut, and the other was smaller than a common pea. These may have originated in colic lymphatic glands, but that was not obvious from their appearance. They appeared to be situated under the peritoneum, and the bowel wall under them was normal. One nodule, similar in appearance, and about the size of a pea was present on the cæcum. All the other abdominal lymphatic glands appeared normal. The gastric mucous

membrane was normal, but beneath the mucous membrane of the small bowel there were half-a-dozen nodules, the largest about twice the size of a barley grain. The mucous membrane over them was intact. Under the mucous membrane of the double colon there were some scores of nodules, and with three exceptions none of these was larger than a barley grain, and most of them were smaller. Many of these nodules had a somewhat linear shape, and some were obviously seated on the course of a small artery. Of the three larger nodules just mentioned, one was as large as a filbert nut, another was a little smaller, and the third was pea-sized. The mucous membrane over the latter showed a distinct defect or ulcer.

The spleen was normal save for one typical glanders nodule on the outer side of its base, under the capsule. It was about the size of a barley grain, yellowish-white in colour, and projected slightly above the splenic surface.

The liver contained a great many echinococcus cysts, the largest about the size of a walnut. All of them had their walls more or less calcified, but the liver parenchyma also contained many solid completely calcified nodules varying in size from a small pea to a pin's head. Nothing bearing any resemblance to a glanders nodule could be found in the organ. The kidneys, bladder, and uterus normal.

The lungs contained a dozen or more of typical glanders nodules, without any marked hæmorrhagic zone. Some had a solid dirty-white central speck, but in at least one the centre was distinctly purulent. No nodules were detectable in the thoracic lymphatic glands. The nose, mouth, throat, pharynx, larynx, trachea, and all the peripheral groups of lymphatic glands appeared normal.

Glanders bacilli were recovered on potato from a lung nodule, the splenic nodule, and from one of the nodules present on the exterior of the colon (cultures not made from the others). The contents of these colic nodules was found to be caseo-purulent. A potato tube inoculated from the nodule on the exterior of the cæcum remained sterile. It was intended to inoculate potato tubes from the three larger submucous nodules in the colon, but when the first one was seared on its outside and punctured, a worm about three quarters of an inch long (S. armatus) escaped with the thick purulent contents. Dissection showed that each of the other two similar nodules also contained a worm.

Two of the submucous nodules in the small intestine and five of the smaller nodules under the mucous membrane of the colon were dissected out with sterile instruments, and triturated in a sterile morter with 6 cc. of bouillon. Of the mixture thus obtained ½ cc. was injected into the peritoneal cavity of each of three guinea-pigs— one male and two females. These animals were killed twenty-one days afterwards, and were found to be quite healthy.

CASE IV.—This case is open to the objection that when the pony was tested with mallein before the experiment was begun there was a rise of temperature amounting to 2·8° F. However, as there was no local reaction, it was thought probable that the animal was not glandered, and it was decided to use it for experiment. As in the other cases, a bolus containing the whole of a potato culture was given by the mouth.

It was tested on the thirteenth day after infection by the mouth

and it reacted both as regards temperature (rise of 3·8°) and the size and duration of the local swelling. It was killed on the fourteenth day after it had swallowed the bolus containing glanders bacilli. The *post-mortem* examination showed the following :—

The mesenteric glands appeared a little enlarged and congested, but on dissection they appeared otherwise normal. The spleen was normal, as was also the liver, save for some echinococcus cysts. The left kidney was normal, but almost half the substance of the right was destroyed by a large abscess containing thick yellow and rather curdy pus. No nodules were discovered in either large or small bowel, and the stomach was normal. The lungs contained about a dozen nodules with the character of glanders nodules, and also one pin-head calcified nodule in one lung under the pleura. All the parts mentioned in the account of the previous cases were examined at the *post-mortem*, but no other lesions were detected.

Glanders bacilli were cultivated on potato from one of the lung nodules. The pus in the right kidney, when examined microscopically, showed great numbers of staphylococci, and a culture of staphylococci with the appearance of a staphylococcus aureus growth was obtained on potato inoculated with some of the pus.

It is not unlikely that this renal abscess was the cause of the elevation of temperature which occurred when the pony was being tested prior to the experiment. Throughout the whole course of the experiment its temperature fluctuated between 100° and 105°, and the test after infection must on this account also be considered uncertain as regards the rise of temperature, though, as previously stated, the local reaction indicated infection.

What, it may now be asked, are the conclusions to be drawn from these experiments? Had they all had similar result to those recorded in Case I., it would have been possible to maintain that they were decidedly against the view that ingestion is the common method of infection in horses, for, contrary to what is usually observed at the *post-mortem* in natural cases, the abdominal lesions in Case I. were conspicuous, or at least so obvious that they could hardly have been overlooked.

In none of the remaining three cases, however, were the lesions in the abdomen conspicuous. In none of these were any macroscopic lesions present in the mesenteric glands, and in Case IV. no glanderous lesions whatever were discoverable in the abdominal cavity, although nodules were present in the lungs. In Case II. the only glanderous lesion discovered in the abdomen was a solitary nodule in the spleen.

Putting aside Case I., it cannot be said that the abdominal lesions in these cases place any great difficulty in the way of supposing that when a horse ingests living glanders bacilli infection may take place, and nodules may develop in the lungs, although no macroscopic lesions are found in any of the abdominal glands or organs. It is true that in three of the four cases abdominal as well as pulmonary lesions were found at the *post-mortem* examination, but there would be no justification for regarding the latter as secondary to the former. It may fairly be said that if there is any difficulty in understanding how glanders bacilli can pass from the stomach or intestines to the lungs without leaving any indication of their course the

difficulty still persists when, as in Case II., one finds pulmonary lesions, and in the abdomen only a solitary small splenic nodule. It is worth noting that in Case II. only six glanders nodules were found in the body, five being in the lungs and one in the spleen, and it would be ridiculous to maintain that the splenic nodule was necessary for the pulmonary infection, or that it might not have been absent.

In my own opinion these experiments make it quite reasonable to maintain that ingestion may be the common natural method of infection in equine glanders, as they in great part remove what has hitherto been the main objection to such a view—viz., the want of correspondence between the lesions in natural cases and in those experimentally set up by causing horses to swallow glanders bacilli.

On the other hand, it is still permissible to believe that horses may be infected by inhalation. In support of the contrary opinion, stress has been laid on two facts, viz. (1) that glanders bacilli are somewhat easily destroyed by dessication ; and (2) the comparative rarity of glanders among grooms and others working in badly infected glanders stables. The first of these objections is not of great weight, for there are no experiments to prove that the degree of dessication necessary to enable a particle of expectorate to float as a dust particle in the air is certainly fatal to glanders bacilli. Besides, as everyone knows, expectorate or nasal discharge in the act of coughing and sneezing may be expelled in such a finely divided form that it immediately floats in the atmosphere, with a possibility that such particles may be inhaled while they are still moist. The second objection is more serious, though it is not fatal to the view that horses are frequently or generally infected by inhalation, for, as the case of foot-and-mouth disease proves, the measure of susceptibility to infection with the same microbe by different channels may be very different in man and the lower animals.

LESIONS.

In my own experience the most constant seat of glanderous lesions is the lung tissue. No case of glanders with lesions elsewhere than in the lungs, and with those organs healthy, has ever been recorded.[1] In nearly every case of farcy, also, glanders nodules are present in the lungs. Strange to say, it is only within comparatively recent years that the almost absolute constancy of lung lesions in glanders has been generally recognised. The explanation of this is to be found in the fact that the word glanders in the older writings on the subject covered only those advanced cases of the disease in which nasal discharge and ulceration of the Schneiderean mucous membrane are prominent symptoms, and attention appears to have been concentrated on these, with the consequence that the less conspicuous but more constant pulmonary lesions were overlooked or not assigned the importance which they deserve.

The ordinary lung lesions of glanders have the form of rounded, firm, or shotty nodules, embedded in the lung tissue, from which they cannot readily be torn or enucleated. The number of these present is very variable, but even in fatal cases of the disease it is not always very great. Hundreds are exceptional, and not rarely the total

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[1] The sentence has been put in this form in recognition of the fact that sometimes in horses which have reacted to mallein no lesions have been discovered anywhere.

number discovered is less than a dozen. When numerous they are usually pretty evenly distributed throughout the lung tissue, but sometimes they are unequally divided between the two lungs. A common size is about that of a pea, but many are smaller, and exceptionally they attain the size of a walnut. When divided mesially they present different appearances acccrding to their age. Recent nodules present a centre of a dirty white colour, and a periphery which is frequently dark red or hæmorrhagic-looking, but sometimes yellowish and gelatinous in appearance. The relative size of the centre and the peripheral part is very variable. Sometimes the former is a mere speck, and at others it amounts to half or more of the nodule. As a rule the central part is not actually liquid, but in a good many cases it has the character of thick pus. In older nodules there is usually no hæmorrhagic peripheral zone, but a greyish or dirty-white opaque central part which may be picked out as a dryish, somewhat crumbling material, and is surrounded by a less opaque tougher part which merges into the surrounding lung tissue. In still older nodules this outer part is distinctly fibrous, and encloses the dry central contents like a capsule. The question whether there is a more advanced stage in which the nodule is completely or almost completely calcified is a disputed one. Glanders nodules certainly appear to have little tendency to calcareous degeneration, but the contention of some authors that calcification never occurs hardly appears to be justified.

When glanders lesions develop in lymphatic glands, or in the spleen or liver, they also take the form of firm nodules, in which one may distinguish a central or a peripheral part, the former being whitish, opaque, and usually rather dry, while the periphery is more translucent, and in older lesions distinctly fibrous. Just as in the case with the pulmonary nodules, those found in other organs sometimes have distinctly purulent centres, and, although the occurrence is highly exceptional, a glanderous submaxillary lymphatic gland may burst spontaneously and discharge pus. The kidneys of the horse appear to have a remarkable exemption from glanderous lesions, and I have only once seen such a lesion in that position.

HISTOLOGY OF THE GLANDERS NODULE.

Two essentially different accounts of the histology of glanders lesions have been given by those who have written on the subject. The first of these describes the foundation of the lesion as a group of cells, similar in appearance to the so-called epithelioid cells which form the bulk of a recent true tubercle (caused by Koch's bacillus). As to what is the source of these cells, opinions are not yet unanimous, but it is generally admitted that they are derived by proliferation from the connective-tissue or other fixed cells of the part, and that, at anyrate, they are not polynuclear leucocytes, and probably not leucocytes at all. This view therefore assimilates the glanders nodule to the true tubercle.

Under the second view the foundation of the glanders nodule is laid by leucocytes which have escaped from the blood-vessels of the part, and are mainly of the polynuclear variety. My own examination of glanders nodules, from all the organs in which they are met with,

convinces me that this is the correct view, at least with regard to the lesions in the horse.

As long as the characters of the cells which compose the central part of a glanders nodule can be made out, it is manifest that these cells are almost entirely polynuclear leucocytes, easily distinguished while undegenerated by the form of their nuclei. What are obviously the youngest nodules met with in the lung are simply more or less spherical areas of the parenchyma in which the air-cells have become crammed with leucocytes. Sometimes, but not always, these air-cells also contain some fibrin, but seldom or never any large proportion of red corpuscles. For a time the walls of the air-cells are quite obvious, forming a net, the meshes of which are compactly filled with leucocytes. Very soon, however, the walls of the pulmonary alveoli which have thus become crowded with leucocytes undergo necrosis and become difficult to distinguish, and a little later they are no longer recognisable. Around this central collection of leucocytes there is usually marked evidence of congestion, especially in the case of the young pulmonary nodules. In these the alveolar capillaries in the walls of the air-cells immediately surrounding those crowded with leucocytes are distended, while the alveolar cavities are usually occupied by a fibrinous exudate or by actual blood.

At its outset a glanders nodule has thus the closest analogy with a miliary abscess, but it would be impossible to confound it with a miliary tubercle. There is also a circumstance that makes it comparatively easy to distinguish between the glanders nodules and the lesions which are determined by any of the bacteria which are typically pyogenic, viz., that the leucocytes and other cells in the former very promptly undergo the somewhat remarkable form of degeneration and necrosis to which Unna has applied the term chromatotexis. This process appears to be essentially a cell necrosis, determined by the glanders bacilli and their products, and its special feature as a necrosis lies in the manner in which it affects the cell nuclei. In other forms of bacterial necrosis the death of the cell is usually not preceded by any marked alteration in the form of the nucleus, and as soon as the cell has lost its vitality the nuclear chromatin no longer stains with the usual dyes. In contrast with this, the nuclei of the cells in the centre of a glanders nodule become disorganised while the cell body is still intact, and the fragments of chromatin resulting from this nuclear disintegration for a considerable period retain a strong affinity for nuclear stains. Hence, in consequence of these persisting nuclear fragments, in sections stained with such dyes the centre of a glanders nodule is dark and opaque, even when necrosis is obviously complete and all structural details have been obliterated. In this process of chromatotexis the chromatin fragments at first assume, for the most part, a spherical form, but when the necrosis is complete and the bodies of the cells have become indistinguishable many of them become elongated or thread-like, with a rounded or club-shaped swelling at one or both of their extremities. Such forms indicate that the altered nuclear chromatin must for a time be in a semi-liquid, plastic condition, and Unna has suggested that the peculiar shapes which it presents are due to the action of the lymph stream. Such irregularly-shaped chromatin particles are, however, found in positions where there cannot be any

actual lymph stream, such as the necrotic centre of pulmonary nodules of quite macroscopic dimensions.

This peculiar degeneration is quite constant in glanders lesions in the horse, and I agree with Unna[1] when he says : " It is not pathognomonic of glanders, it is found also in other necrotic processes, though never in such high degree and so invariably as here." In the necrotic centres of true tubercles exactly similar chromatin fragments which have retained their staining affinities are sometimes met with, but the chromatotexis seen in tuberculous lesions is distinguished from that of glanders by the fact that in the former the necrosis does not ensue so promptly, and is very soon followed by complete disintegration of the nuclear chromatin, which ceases to stain with nuclear dyes. In glanders lesions, on the other hand, this peculiar degeneration sets in almost immediately, but the nuclear fragments are remarkably persistent, since they are found in nodules whose fibrous capsules indicate that they are of old standing.

I have been most struck with the importance of this form of necrosis as an assistance in the recognition of glanders lesions by observing that it not only occurs in the ordinary circumscribed nodular lesions, but is also a marked feature in acute diffuse glanderous pleurisy. In such cases the layer of fibrinous lymph on the surface of the inflamed pleura contains numerous polynuclear leucocytes, which are here and there aggregated into clusters like miliary abscesses. Similar collections of these cells are met with in the same position in the much commoner cases of streptococcus pleurisy in the horse, but the two lesions are easily distinguishable histologically by the fact that pronounced chromatotexis of the leucocytes occurs in glanderous pleurisy, while it is entirely absent in the other. Unna,[2] in his account of the histology of the glanders nodule of the human subject, says, (1) "it is striking in its homogeneity," and (2) "nor is there any intimation of a local leucocytosis ; the bacilli appear to be chemotactically indifferent to wandering cells." Precisely the opposite is the case in glanders nodules in the horse. These are practically never homogeneous, but have a central area which is different in appearance from the periphery, and there is the most striking evidence that glanders bacilli are not chemotactically indifferent to the wandering cells, since the centre of the nodule is always an aggregation of polynuclear leucocytes. This is no secondary invasion of the nodule, but constitutes its actual foundation. Besides, it may be observed that it is possible by a very simple expedient to obtain the most convincing proof that the polynuclear leucocytes of the horse have a tendency to ingest glanders bacilli. When living and virulent glanders bacilli are mixed with warm citrated blood of the horse (drawn off from the jugular by means of a hypodermic syringe) more or less pronounced phagocytosis takes place in a few minutes, and in the case of some horses (non-glandered) one may find as many as twenty bacilli in a single leucocyte. It may be added that apparently only the polynuclear variety have this tendency. This, of course, cannot be put forward as proof that the young glanders

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[1] "The Histo-Pathology of the Diseases of the Skin," English translation, p. 462.
[2] *Loc. cit.*

nodule is almost exclusively composed of leucocytes, but it is a fact
entirely consistent with that contention.

The lack of homogeneity on the part of glanders nodules in the
horse is due to the fact that when they are quite recent they have
(at least in the case of the lung) a peripheral zone marked by the
effusion of a fibrinous exudate or of actual blood, while when they
are older the leucocyte centre is surrounded by cells of a different
kind. In its simplest form the outer part of these older nodules is
a thin zone of young fibrous tissue. This tissue is most fully formed
(possesses most matrix) in its outer part, and inwardly, where it
meets the degenerated centre of the nodule, it may be composed of
fibro-blasts without any intercellular substance. Between the outer
distinctly fibrous capsule and the margin of the degenerated centre
there may be a zone, occasionally of considerable breadth, composed
of rather large cells with pale-staining nuclei and exactly resembling
the so-called epithelioid cells of a true tubercle. When giant cells
are present in a glanders nodule they are ordinarily situated in this
zone, but they may be found further out, where some of the fibro-
blasts have already begun to form a fibrous matrix. Giant cells may
be absent from glanders nodules even when these are obviously of
some standing, but in the majority of not quite recent nodules giant
cells are present, although the contrary has been stated. Moreover,
the giant cells of glanders nodules are quite indistinguishable from
those which are more constantly and abundantly present in tuber-
culous lesions. An important difference between the two kinds of
lesions is that giant cells are never present in the central part of
glanders nodules, whereas they are found indifferently in all parts of
miliary tubercles.

To sum up this part of the subject, it may be said that the glanders
nodule of the horse, so far as regards its histology, is a lesion *sui
generis*. It has only a distant resemblance to the true tubercle, and
it would scarcely be possible to confound them. On the other hand,
it has a distinct affinity with the miliary abscess, especially in its
early stage, since in each case the lesion is an area from which the
normal elements have disappeared while their place has been taken
by leucocytes. However, there are two features which distinguish
the glanders nodule from the miliary abscess. The first is the
peculiar degeneration (chromatotexis) which never fails to set in in
the central part of a glanders nodule, and the second is the slight
tendency to peripheral extension which the glanders nodule ordinarily
exhibits. In a miliary abscess the size of the lesion is limited only
by the life of the animal, and these lesions are ordinarily small
simply because pyæmic conditions are soon fatal. Glanders nodules,
on the other hand, soon come to a standstill, the initial lesion having
had the effect of confining the bacilli and preventing a progressive
invasion of the surrounding tissue. In exceptional cases, however,
where the usual balance between the virulence of the bacilli and the
protective powers of the animal organism is not present, the glanders
nodule may continue to extend, while its central part undergoes
liquefaction. In these cases the analogy between glanders lesions
and those caused by the habitually pyogenic bacteria becomes very
close.

If one leaves out of account the peculiar degeneration which over-

takes the central part of a glanders nodule, that lesion also presents a close histological affinity with the granuloma of actinomycosis. In that also the foundation of the lesion, and the part of it which immediately surrounds the parasites, is almost entirely composed of leucocytes. There is also observable the same tendency to an early arrest of the growth or extension of the lesion, while the fibroblasts or other fixed tissue cells begin to construct a fibrous or epithelioid zone around the leucocyte centre.

The lesions of farcy are at the outset histologically identical with the solid lesion which is usually termed a glanders nodule. There is the same migration of leucocytes, and the same disappearance of the normal tissue elements from the parts where the leucocytes have congregated. But what is only exceptional in the case of the pulmonary nodules—the progressive liquefaction of the tissues around the initial lesion—is the rule in the lesions of farcy, which thus become converted into actual abscesses. The subcutaneous tissue, perhaps in consequence of its richness in lymphatic channels, appears to be a specially favourable soil for the glanders bacillus, and one in which its tendency to progressively invade the tissue around the initial lesion is less easily held in check by the leucocytes. Here also the leucocytes are liable to chromatotexis, but that is much less complete than in the centre of the solid glanders nodule. As is well known, the pus which is discharged from a farcy bud is ordinarily less opaque and creamy than that which is formed under the action of the common pyogenic bacteria. Its greater viscidity and slighter opacity are apparently due to an unusual proportion of albuminous material and a smaller number of leucocytes or pus corpuscles. There are, however, farcy lesions in which the pus has nothing in its naked eye characters to distinguish it from the pus of what may be called ordinary abscesses.

SOME DISEASES COMPLICATING RINDERPEST AMONG CATTLE OF INDIA.

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RINDERPEST is a disease which runs a very regular course, and one of which the diagnosis usually presents little difficulty. Among the cattle of India the degree of susceptibility to this disease varies very considerably. In some districts in the Plains, where the ravages of rinderpest are of frequent occurrence, the percentage of animals resistant to the disease is very high, and in many outbreaks a mortality of about 25 per cent. only has been recorded. This immunity may be ascribed to such causes as "hereditary influence," protection afforded by a previous attack of the disease, and survival of the fittest. In other parts of the country, especially in the mountainous tracts, where traffic is less, and cattle are not exposed to the danger of infection in fairs and markets, rinderpest is almost unknown, but when it does make an appearance it carries off about 90 per cent. of animals attacked. In the first class of animals, namely, the bulls and cows ordinarily met with in the villages of the plains, the course of the

disease is somewhat modified, the stages are more prolonged, the symptoms are less marked, and recovery is frequent.

In the cattle of the hill breed the disease takes a very acute and fatal course. In inoculated cases the three stages of the malady are well marked, and each is usually of about three days' duration. These are (1) the incubative period, (2) the febrile stage, when all the lesions can be observed, and (3) the stage of diarrhœa, on the second day of which the temperature rapidly falls, and the animal dies on the eighth or ninth day.

The *post-mortem* appearances in every case show a striking similarity, and in this country secondary lung lesions are rarely found. The pathological changes throughout the whole mucous membrane of the alimentary canal present a picture which is not simulated by any other disease. There is, perhaps, no solitary lesion upon the existence of which a certain diagnosis can be founded. Before general exfoliation of the mucous membrane takes place, the pin-point ulcers on the surface of the tongue, gums, and pharynx are typical. The formation of vesicles and ulcers on the mucosa of the gall bladder is stated by some authors to be characteristic of rinderpest alone. These lesions, however, have been observed in fatal cases of Texas fever, and I have seen the same appearances caused by the presence of distomata. The congested condition of the Peyer's patches often exists in other inflammatory affections of the bowels, but in no other disease of cattle are these glands found to be covered with necrotic material as in rinderpest.

The typical case of rinderpest is one which it is not difficult to recognise, and anyone who has had an experience of one or more outbreaks may consider that there is little more to be learnt of the clinical or *post-mortem* symptoms.

There are, however, many variations in both, due not to the disease itself but to other pathological conditions playing a secondary part. When an isolated or individual case of such a kind is met with it is very frequently a matter of difficulty to form a decided opinion. I have seen many animals die from the disease which during life showed no characteristic symptoms. At the *post-mortem* all the typical necrotic changes in the alimentary canal were marked. Many cases show a rise of temperature only, and after death there is nothing to be found which might not be due to gastro-enteritis.

Many causes may be held to account for these phenomena : a variation in the virulence of the contagium, a difference in the resistant power of the cells of the body, or the still less understood term of individual idiosyncracy.

There is no doubt that these and other unknown factors cause many deviations in the typical course of a disease.

It has, however, been observed in this and in other Laboratories where anti-rinderpest serum is prepared that certain other diseases frequently run a course concurrent with rinderpest, and in many respects either intensify or modify the effect of that disease. The recognition of such secondary affections, and an understanding of the part played by them, is of much importance both in the preparation of serum and in its practical application.

Some of these diseases I have had an opportunity of studying at the Bacteriological Laboratory, Muktesar, among the animals experi-

mentally inoculated with rinderpest virus, for the purpose of preparing the anti-serum, and for testing the potency of such serum before issuing.

PIROPLASMOSIS.

This scourge of cattle, known under many names (Texas fever, redwater, bovine malaria, tristeza, etc.), is one which has been recognised in almost every country and fully described by many authors.

In India the disease is said to have been first clinically observed by Hallen in 1871, but from later publications by Queripel, Gunn, and others, and from regulations laid down concerning the examination of horses imported from Australia to prevent ticks being brought in, the existence of Texas fever in the country does not seem to have been accepted as proved.

Queripel[1] discussed the question, and was inclined to believe that the disease did exist in India, as the symptoms of "red urine" and "wasting" were well known to zemindars. At the same time, he advised that horses landed from Australia should be examined for ticks.

Gunn[2] stated that all microscopical examinations of blood had failed to demonstrate the micro-organism of Texas fever, and expressed the view that the ticks of India were not contaminated with the parasite of the disease. He concluded that the disease did not exist in India, and that all precautions should be taken to exclude it from the country.

In 1898 Raymond reported the presence of piroplasma bigeminum in cattle of India.

The piroplasma was demonstrated in the blood of the cattle of this country by Stockman[3] in 1903, while conducting some experiments relative to the degree of susceptibility to rinderpest among breeds of cattle in the Madras Presidency. Ten out of twenty-three cattle inoculated with rinderpest developed redwater.

The typical pear-shaped bodies are found in the red cells of affected animals. The disease is apparently the same as observed in other countries, and is spread by means of ticks. At the present time we are aware that the protozoa are harboured in the blood of a large percentage of cattle, but we have no accurate knowledge of the extent to which the disease by itself affects animals. Information collected from native owners of cattle shows that a disease recognised by the symptoms of red urine is known to them, and that at times it causes considerable loss. During last year Captain Walker, I.C.V.D.,[4] reported an outbreak of Texas fever where eight animals died.

In the blood of many cattle apparently healthy and in good condition, the small coccus-like bodies described by Smith and Kilborne have been found in the red cells, while a careful and extended search did not reveal any other form of the parasite. It is a significant fact that the pear-shaped bodies and bacillary forms characteristic of the piroplasma bigeminum have been found only in the blood of cattle after they have been inoculated with virulent rinderpest blood.

1 " Agricultural Ledger Series," No. 2 of 1897.
2 " Agricultural Ledger Series," No. 2 of 1899.
3 " Veterinary Record," 11th April 1903.
4 Report of Civil Veterinary Department, Punjaub, 1903.

This seems to point to the conclusion that the cattle of India have for many years had this disease among them, and that a large percentage of animals now possess an immunity sufficient to protect them until this resistant power has been reduced by an attack of another disease. It would also seem as if rinderpest more than any other disease broke down this natural resistance to piroplasmosis. I have made many examinations of the blood of bulls whose vitality had been lowered by an attack of anthrax or black-quarter, or by large subcutaneous injections of virulent cultures of these diseases ; also of cattle dying from gastro-enteritis, pneumonia, and other affections, but have not in such cases found the blood infected with the malarial organism.

In other countries it has been noted that cattle inhabiting a region over 5000 feet high do not suffer from malaria, and the reason ascribed is that ticks do not live at this altitude. This is not the case in India. In the Kumaon tract of the Himalayas, at a height considerably over 7000 feet, the cattle are infested with ticks, and I have found a larger percentage of these animals affected with piroplasms than among cattle of plains breed. The parasite found in the blood of these hill bulls presents certain peculiarities.

It is generally seen in form of a spore, ring, rod, or leaf, with a chromatin dot at the apex. I have not been able to find any of the typical pear-shaped forms, and I have not observed the blood-coloured urine which is usually a clinical symptom of the disease in cattle of the plains. In hill cattle when the piroplasma appears numerous in the blood, during the course or after recovery from a modified attack of rinderpest, a fatal issue almost invariably occurs. In plains cattle the complication has not, as a rule, so rapid and fatal an effect ; some cases recover, and the micro-organisms after a time disappear from the blood. It is a question whether, when an animal becomes infected with piroplasms after a subcutaneous injection with virulent rinderpest material, the organism was introduced with the inoculated blood, or had been previously lying dormant in the body. I am of opinion that the latter is more frequently the correct view. In many instances I have examined the virulent blood before inoculation without being able to find any trace of organism, and subsequently the piroplasms have appeared in large numbers in the blood of the injected bull. Also, I have many times injected, both subcutaneously and intravenously, fresh blood containing numerous parasites into a healthy bull, and in no instance have I been able to produce the disease.

As a complication of rinderpest, piroplasmosis appears from five to fifteen days after the inoculation of virulent blood. The only clinical symptoms which at first give a clue to the presence of the disease are, fluctuating temperature, with considerable variation between the morning and evening record, an anæmic condition, and an apparent relapse in animals which are recovering from an attack of rinderpest.

The following cases show the general course of the disease :—

Bull No. I.—Plains breed. Received a simultaneous injection of serum and virulent blood on 28th March, with the object of immunising him for the preparation of serum. He had a high temperature reaction, with the appearance of vesicles and ulcers on the tongue and buccal membrane. On 12th

April the temperature had returned to normal and the animal was feeding well. On 15th April, the temperature again suddenly rose to 40° C., and from this date fever was continuous, with a difference of 2° to 3° C., between morning and evening temperature. From the appearance of fever the blood was examined daily. Numerous pear-shaped organisms were observed in the red cells. The parasites after a week gradually disappeared, until only a few coccus-like forms could be found. About the end of April the bull was in good health again. He was subsequently injected with 6000 cc. of virulent rinderpest blood and afterwards frequently bled for serum, but did not again develop any symptoms of illness.

Bull No. II.—Plains breed. Was injected by the simultaneous method on the same date as bull No. I. He had a severe attack of rinderpest lasting seven days. About the tenth day the lesions were healing and the bull was recovering, when the temperature again rose to 39.5° C., and intermittent fever continued for some days. The blood was examined, and about 30 per cent. of the red cells were found to contain the piroplasms. The animal became daily more emaciated, fed little, coat staring, urine blood-coloured, diarrhœa constant. The condition became worse, and the bull died on the twenty-fifth day.

At the *post-mortem* the yellow connective-tissue ecchymoses and extra-vasations, enlarged and softened spleen, and other lesions characteristic of Texas fever, were marked.

In the cases observed of bulls of hill breeds affected with piroplasmosis in conjunction with rinderpest, the symptoms were much the same, with the exception that the fluctuating temperature and the piroplasms in the blood appeared at an earlier date, and the disease ran a more rapid and fatal course.

Bull No. III.—Hill breed. Received a simultaneous injection of serum and virulent blood. On the fourth day the evening temperature was 40.2° C. No symptoms of rinderpest appeared, but fever continued, with 2° to 3° C. variation between morning and evening temperature. There was a serous discharge from the nostrils, and the mucous membrane was of a yellowish colour. The animal fed little, continually lying down and at times struggling. Died on the ninth day. The blood was examined on the fifth day, and numerous piroplasms were found. They were of small size, the majority in the shape of a leaf with a chromatin dot at the point, but many were in the form of a ring and others were rod-shaped. Before death the micro-organisms increased somewhat in size and became more numerous. No pear-shaped forms were found. At the *post-mortem* no lesions of rinderpest were observed. The connective-tissue and mucous membranes were of a yellow colour, and there were small sub-capsular hæmorrhages in the spleen and liver. The spleen was enlarged and softer than normal. Liver engorged. Ecchymoses on surface of intestines, with areas of deep congestion.

Bull No. IV.—Was inoculated in similar method as above. He had a slight attack of rinderpest, and was recovering, when, on the twelfth day, the temperature again became high, and it remained fluctuating for some days. The condition of the animal each day became worse; appetite failed. anæmia was marked, diarrhœa set in, and death occurred on the nineteenth day after inoculation. The blood was examined on the twelfth day and piroplasms were found similar to those seen in the blood of bull No. III. They became slightly larger and much more numerous as the disease advanced. At the *post-mortem* scars of healed ulcers in the small intestines, pitted Peyer's patches, and other signs of a recent recovery from rinderpest were noticed, together with the characteristic appearance of death from Texas fever.

Piroplasmosis as a complication of rinderpest has been found at this laboratory to be a cause of considerable loss and trouble. At times it carries off bulls which have been immunised, and which have received a large injection of virulent blood for the preparation of serum. Among the animals inoculated with virulent material for purpose of producing the disease and obtaining blood for injecting serum-making bulls, piroplasmosis frequently caused a variation in the temperature curve and ordinary course of rinderpest. Such animals are not considered suitable for bleeding, and are wasted.

When the potency of a bulk of anti-rinderpest serum is being tested by inoculating a number of bulls with virulent blood and varying doses of serum, the appearance of Texas fever increases the difficulty of ascertaining the amount of serum required to protect against rinderpest, and necessitates the repetition of the experiment.

In the practical use of the serum in outbreaks of rinderpest it is probable that Texas fever arising as a secondary disease may play an important part, and may account for a certain percentage of deaths. Up to the present no observations in this point have been recorded in India.

TRYPANOSOMIASIS AMONG CATTLE OF INDIA.

In a communication which appeared in the preceding number of this Journal (p. 209), a trypanosoma found in a smear preparation taken from the spleen of a hill bull was described by Capt. Durrant, A.V.D., and myself. As recorded in the above paper, the bull had been used for the purpose of testing the potency of a bulk of anti-rinderpest serum. He had a slight attack of the disease, from which he was recovering when symptoms of a secondary disease became manifest: diarrhœa, fluctuating temperature, loss of appetite, staring coat, anæmia, resulting in death.

Since the first discovery of this trypanosoma, I have met with many more cases of trypanosomiasis among the cattle of hill breed kept at this Laboratory for experimental purposes, and consequently have had an opportunity of a fuller study of the parasite. In every instance in which the disease has been observed, it has made its appearance secondarily to an attack of rinderpest, or after the animal has been inoculated with a large quantity of virulent blood in order to hyper-immunise for the preparation of serum. The following are the notes of three cases :—

Bull No. I.—Hill breed. Was immunised by a simultaneous injection of virulent rinderpest blood and serum. He had a slight attack evidenced by temperature reaction only. Fourteen days after the first inoculation he was injected subcutaneously with 2500 cc. of virulent blood for the purpose of hyper-immunising. This was followed by a rise of temperature, which remained near 40° C. for six days and then returned to normal. After three days the temperature again reached 41° C., and stood at that for eight days. On the ninth day the temperature rapidly fell, and the animal died the following night. During the last ten days the bull ate little, became weak and emaciated, and was continually lying down. The last day the animal struggled convulsively; some serous fluid flowed from the nostrils; mucus and blood from rectum. On the appearance of secondary fever the blood was examined and trypanosomata found. On the first day of observation

there were four parasites in a field, but the number daily increased, and at the time of death they numbered thirty in a field.

Bull No. II.—Hill breed. Was used for the purpose of serum-testing. At the time of inoculation he was in good condition. He had a fairly severe attack of rinderpest, which lasted about twelve days. The lesions of this disease healed, and the temperature returned to normal. The bull, however, remained thin, did not recover its former strength, and suffered from diarrhœa. The temperature was irregular, and frequently rose above normal. The blood was examined and trypanosomata discovered. They, were not numerous, varying from one to three in a field. The animal died a month after the date of inoculation. At the *post-mortem* of this bull there were no marked lesions ; the carcase was emaciated, the blood watery, the spleen somewhat enlarged and soft, and petechial submucous hæmorrhages in small and large intestines.

Bull No. III.—Hill breed. Was hyper-immunised for serum-making, and received a subcutaneous injection of 3000 cc. virulent rinderpest blood. This was followed by a rise of temperature, which remained above 40° C. for some days, after which the chart showed a very fluctuating curve, with a difference of 2° to 3° C. between the morning and evening record. The blood was examined for piroplasms and found to contain trypanosomes, two in a field. After several days the temperature became normal, the trypanasomes disappeared, and the bull recovered.

My further observation of the trypanosomes has shown that the parasites which were first noticed, and which were taken from the spleen, were evidently immature forms, and that the description then given differs very considerably from that of the mature flagellate form found in the blood stream.

The full-grown trypanosomes are much longer than the surra parasites, and are probably the longest yet described. The largest depicted in the accompanying Plate measured 91 μ in length. The average length is from 60 to 80 μ. The breadth is from 2 μ to 4 μ, and in some cases as much as 6 μ ; the latter forms were not typical, and seemed to be undergoing change. The posterior extremity is very fine and elongated, measuring from centrosome to end 10 to 17 μ. The flagellum is well developed, and measures from 15 μ to 25 μ. In the younger forms the centrosome is situated very close to the nucleus. In the blood taken from two infected bulls I found peculiar developing forms bearing a striking resemblance to the crescents of human tertian fever. These are large crescent-shaped bodies staining blue and containing numerous chromatin granules.

As I have stated above, the disease was in every instance found in cattle of hill breed which had been treated with rinderpest virulent blood. Up to the present I have not been able to find the trypanosoma in the blood of cattle of plains breed or in uninoculated hill cattle. The apparent immunity of plains cattle may be due to the fact that these are all specially selected cattle, in good condition. This is not the case with the hill bulls ; they are of all kinds and conditions, and many are emaciated and half-starved animals. The relation between the rinderpest inoculation and the appearance of the trypanosoma appears to me similar to that observed in piroplasmosis of Indian cattle. These animals, more especially those of certain districts, have probably been for many years subject to the attack of these parasites, and now possess a partial immunity. The disease lies dormant in the body until the normal resistant power of the cells

has been lowered from some cause, such as an attack of rinderpest. The parasites then develop in the system, exercise a certain amount of harmful effect, and assist the primary disease to a fatal issue.

There is another way in which the appearance of the trypanosoma after rinderpest inoculation may be accounted for. It is possible that the trypanosoma may be conveyed from an infected to a healthy animal with the rinderpest virulent blood. This latter view is less probable. The plains cattle which have been injected with a large dose of virulent blood from hill cattle have not become infected with the trypanosoma ; neither have I been able to convey the disease to any experimental animals by means of either subcutaneous or intra-venous injections of blood containing numerous trypanosomes. The disease is frequently associated with piroplasmosis.

In the blood of the bull from which the trypanosomes shown in Plate VIII. were taken about 50 per cent. of the red cells were invaded by small piroplasmata. Some cells contained four parasites. This case ended fatally. The disease does not show itself by any distinctive clinical symptoms. In most of the animals in whose blood the trypanosomes were found the existence of the disease was not suspected until the blood was examined. In some instances attention was drawn to these bulls by their poor and anæmic condition. In other cases the bulls at first were in apparent health, but their daily chart showed a fluctuating temperature, with an abnormal variation between the morning and evening record.

Only a few cases ended in death, and these bulls were in debilitated condition. In others the trypanosomes were found for several days in the blood, never very numerous, seldom above five in the field. They subsequently disappeared, and were not discovered again, though the blood was examined daily for five to six weeks.

The following experimental inoculations were carried out, but I failed to transmit the disease in so far that the trypanosomata was never found in the blood of the inoculated animals :—

Pony No. I.—Inoculated subcutaneously with 25 cc. defibrinated blood from Bull No. 3276. In the blood used from Bull No. 3276 there were four parasites in a field.

Pony No. II.—Received intravenously 50 cc. citrated blood from Bull No. 3025. In this blood there were six trypanosomes in a field.

Bull No. I. (Plains breed) ⎱ Were inoculated subcutaneously with 25 cc.
Bull No. II. (Hill breed). ⎰ defibrinated blood from Bull No. 3276.
Bull No. III. (Plains). ⎱ Were inoculated intravenously with 50 cc. citrated
Bull No. IV. (Hill). ⎰ blood from Bull No. 3025.
Rabbit No. I.—Received 5 cc. defibrinated blood from Bull No. 3276.
Rabbit No. II.—Was inoculated intravenously with 2 cc. citrated blood from Bull No. 3025.

ECHINOCOCCUS DISEASE.

This disease is very prevalent among the cattle of India. Pease collected statistics, and found that, among the cattle slaughtered for food in Lahore, 80 per cent. were affected to a greater or less degree. He states from his own experience that hydatid disease is a common cause of death and loss of efficiency among cattle and camels. The prevalence of the disease is due to the number of pariah dogs and jackals, and the defective method of the disposal of the carcases of

dead animals. At the Laboratory I have noticed that bovines badly affected with hydatids succumb very quickly to an attack of rinderpest. Frequently, after having been inoculated, the animal dies in the incubative stage, before the lesions of rinderpest have appeared. In other cases the temperature does not rise on the third or fourth day, but continues abnormally low, gradually falling until death results on the fifth to seventh day.

In these animals the course of rinderpest is always erratic, and the characteristic symptoms either do not present themselves at all or appear irregularly. At the *post-mortem* the lesions of rinderpest are rarely well marked, and at times altogether absent.

Bull No. I. Hill breed.—Received a simultaneous injection for the purpose of testing serum. On the second day the morning temperature was 40·4° C., fell below normal on the following day, and remained low. The animal died the next day. There were no other symptoms of illness. The lungs were crammed with cysts, varying in size from a cricket ball to a marble. The liver was also affected, and the spleen contained one cyst about the size of a cocoanut. The mucous membrane of the small intestines was bile-stained and covered with mucus. No lesions of rinderpest were noted.

Bull No. II. Hill breed.—Was inoculated with virulent blood as a control. The temperature remained normal during the first four days. On the fifth it reached 40·5° C., and on the next day fell to 36·3° C., the bull dying on the following day. The lungs and liver were the seat of numerous cysts. The gall-bladder was absent, and in its place was a small cyst the size of a marble containing clear fluid. Another cyst the size of a pea-nut was situated on the border of the central flap of the tricuspid valve of the heart. There were a few vesicles on the gums and under the tongue ; the intestines showed slight congestion, but there were no further lesions of rinderpest.

Bull No. III. Hill breed.—Was used for serum-testing purpose, and received a simultaneous injection. On the second and third days the temperature was 39·6° C. On the fourth day it fell to 37° C., and remained low with slight variation for five days. On the tenth day the temperature was 36° C., and the bull died on the eleventh day. The animal had fed well until two days before death, when the appetite failed and the bull remained lying down. There were no symptoms of rinderpest. The lungs, liver, and spleen contained numerous cysts, and there was congestion of the mucous membrane of the fourth stomach and small intestines. The Peyer's patches were raised and congested.

During the year I observed about thirty cases of a more or less similar kind, where the symptoms of rinderpest were suppressed, and few lesions beyond the presence of numerous cysts to be found after death.

DISTOMATOSIS.

This disease is known to be prevalent among cattle grazing in marshy land subject to inundations. Pease has given a description of the disease as met with among India cattle, and of the parasite, the fasciola hepatica. In its relation to rinderpest exactly similar observations have been made as described in cattle affected with hydatids. The temperature seldom rises, tends to go below normal, and the animal dies in the early stage, or, if it lives a longer period, no marked symptoms of rinderpest appear. At the *post-mortem* it is often difficult to say what the cause of death has been. Beyond the

cirrhotic liver, congestion of the fourth stomach and intestines to a greater or less degree, and sometimes a bile-stained appearance of the tissues, there were no changes of sufficient severity to account for death.

Animals affected with hydatids often remain in apparent health and good condition, but those suffering from distomatosis are generally emaciated and anæmic, skin dry, and bowels irregular, and this state renders them more susceptible to the attack of any other disease.

APHTHA.

During the winter months a contagious form of aphtha frequently makes its appearance among the cattle kept at the Laboratory for experimental use. The infection is brought by the hill cattle coming from the parts some 60 to 80 miles further in the Himalayas. The disease affects the muzzle, angles of the mouth, mucous surface of gums, lips, under surface of the tongue, and floor of the mouth. It is contagious and spreads very rapidly among the whole herd. It causes no elevation of temperature, and only affects the animal in so far that the lesions of the mouth render feeding painful, with a consequent loss of condition. The sores heal and the effect of the disease passes over in about three weeks. In the first stage vesicles are formed which exude a serous fluid, and these change into ulcers which become covered by a crust and take several days to heal. Animals suffering from this form of aphtha are less resistant to an attack of rinderpest, owing to their debilitated state. It is also frequently difficult to distinguish between the aphthous ulcers and those of rinderpest, when situated in the mucous membrane of the mouth. The ulcers of aphtha tend to run together into a circular lesion about the size of a threepenny piece or a sixpence. They are also covered with a necrotic crust. In rinderpest the ulcers are clear-cut and pin-point in appearance, do not run together, and are not covered with crust.

DESCRIPTION OF PLATE VIII.

a. Trypanosomata found in blood of cattle.
b. Forms of the same in smears from the spleen.
c. Piroplasmata in red blood corpuscles.

Drawn with Zeiss oil-immersion apochromatic 2 mm. lens. Compensating ocular 8. Magnification 1000 diameters.

THE PROPHYLACTIC TREATMENT OF RINDERPEST BY MEANS OF PREVENTIVE INOCULATION, MORE ESPECIALLY CONSIDERED IN REGARD TO THE CONDITIONS PREVAILING IN INDIA.

By Captain G. K. WALKER, F.R.C.V.S., Indian Civil Veterinary Department.

IT may be well, at the outset, to say a few words with regard to the peculiar conditions appertaining to the treatment of cattle disease in India. It is a matter of fairly common knowledge that cattle are popularly regarded as sacred in that country. This assertion serves
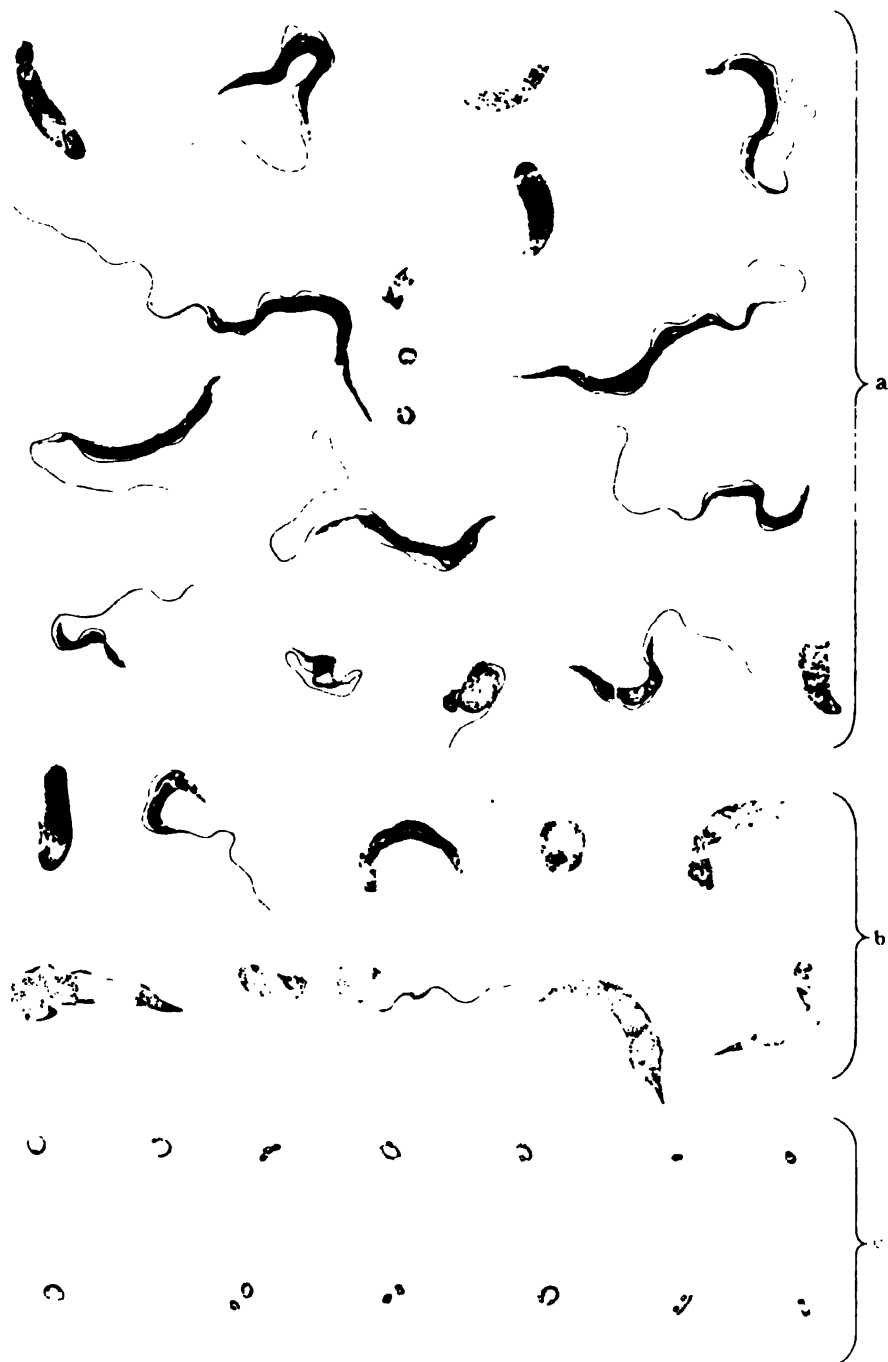
to describe a condition of things very antagonistic to strong prophy-
lactic measures. It may be said that the vast majority of the
natives of India regard with great repugnance the slaughter of cattle
in any shape or form. Frequently they object to the performance
of *post-mortem* examinations, and in some cases to minor operations.
As a rule they are very apathetic, and a visitation of cattle plague is
regarded from a purely fatalistic point of view. In severe outbreaks,
however, it may happen that some of the more intelligent are not
content to remain indifferent. In these cases the native treatment
usually takes the form of some weird religious ceremony, from which
some religious mendicant materially benefits. Passive, and occasion-
ally active, opposition is usually displayed if Western preventive
methods are attempted.

In spite of all this there are signs that preventive inoculation, with
serum alone, against rinderpest is becoming popular. In the Punjab,
where there is a large Mahomedan element, a large number of
cattle have been successfully inoculated in actual outbreaks, and in
other parts of India, and also Burma, inoculations have been carried
out. The success and growing popularity of vaccination against
small-pox in human beings has been a factor in paving the way.
In course of time natives may report outbreaks of disease and ask for
assistance. This is occasionally done now, but as a rule outbreaks
are concealed. European cattle-owners generally ask for help, as
might be expected. Cattle owned by Europeans are in quite an
insignificant proportion, and need not be considered when discussing
methods of prevention applicable to India generally. It is always
possible to deal with each outbreak on estates belonging to Europeans
on its own merits, in consultation with the persons concerned.

Rinderpest, as it appears in India, differs in many respects from the
disease as seen in Western Europe or South Africa. It has, no
doubt, existed there from time immemorial, and it is said to be
closely comparable to the disease as met with in the Steppes of
Russia and in Turkey. So far as the actual symptoms are concerned,
there is probably no great distinction to be drawn. It is worthy of
note, perhaps, that in India skin lesions appear to be uncommon, and
the lesions in the mouth are probably rarely so severe as those seen
in Great Britain, if the models exhibited of the disease, as it appeared
in the latter country, are accurate.

The disease in India is endemic, and in the plains the mortality
among animals attacked is not large compared with that experienced
in South Africa and Great Britain. From a large number of figures
it is found that the average mortality in the plains of India is from
40 to 50 per cent., but in the Himalayas it is about 90 per cent.

Wide variations in mortality are met with in different parts of
India, and even in neighbouring villages. The virulence of the
disease and the immunity of the cattle vary to such a degree that it
is practically impossible to fix doses of certain preventive materials,
when such a proceeding is indicated, except after experiment upon
the scene of the outbreak. This has rendered the application of certain
methods of preventive inoculation a complicated and difficult matter.

The virulence of the disease is usually greatest in the cold season,
and it is always most marked at the commencement of an outbreak.
Nevertheless, outbreaks occur in the hills without any apparent

decrease in the virulence of the disease. It is obvious that this variation in virulence is explained to a great extent by the fact that the most susceptible animals are sure to be first attacked, but some observers are strongly of opinion that the virus has a tendency to become attenuated towards the end of an outbreak.

The few facts recounted above will show that in instituting a method of preventive inoculation for rinderpest in India many points had to be taken into consideration, and that there were many difficulties to be surmounted. In addition, many other objections to energetic measures existed. They were chiefly financial and political, and need not be discussed in this paper. Before inoculation work was instituted in India much had been done in South Africa and other places, and some of the methods could be regarded as having gone beyond the experimental stage. It was thought advisable, however, to make a series of experiments both in the laboratory and in the field, for reasons given above. Experimental work in India with regard to preventive inoculation against rinderpest dates from 1897, when Professor Koch gave a demonstration at Muktesar laboratory on his way back from South Africa.

From that date preparations were made for experiments on a large scale. Drs Lingard and Rogers are the principal names connected with the laboratory experiments, and they and certain officers of the Indian Civil Veterinary Department are responsible for the field operations.

The different methods of preventive inoculation will now be put forward in turn.

The routine of each method will be explained and its applicability to Indian conditions discussed. Methods of inoculation practised before 1896 will be passed over. They were crude, and consisted in direct transmission of the virus of rinderpest from animal to animal by various means. An exception to this statement may be made in regard to the experiments of Semmer in Russia in 1895. This observer ascertained that the blood of an animal that had recovered from rinderpest exercised an immunising influence if injected into a healthy animal, and Pitchford and Theiler confirmed this in South Africa in 1896. Severe losses from rinderpest in South Africa resulted in Professor Koch visiting that country in 1896, and protective inoculation on a large scale may be said to date from that time.

I.—KOCH'S BILE METHOD.

In a series of reports to the Secretary for Agriculture, Cape Town, Koch[1] described a number of experiments that he had conducted. The greatest prominence was given to a method of preventive inoculation with the bile from an animal suffering from rinderpest. It was one of the earliest methods instituted in the field, and, until superseded by others, popular, and considered hopeful. Koch does not seem to have stated anywhere why he tried bile as a protective agent. In his second report, however, when discussing a ready method of transmitting the virus of rinderpest from one animal to another, he says : " Experiments were also made with bile which was taken from animals that had succumbed to rinderpest, and which was injected in the subcutaneous tissue. The motive for

so doing was because a mixture of bile or other liquids was said to be sometimes used by Free State farmers, and also the circumstance that in the bile of most of the cases examined I have found, in pure cultivation, a bacterium which, according to the description published, is conformable with the microbe discovered by Dr Simpson in Calcutta, and declared to be the cause of rinderpest."

These experiments with bile had negative results, and Koch went on to say in the same report: "We are consequently justified in saying that bile does not contain the contagion of rinderpest, and that Simpson's bacterium cannot be considered to be the microbe of rinderpest."

The above statements are important in view of the question of the kind of immunity conferred by bile inoculation. Probably these experiments led Koch to try bile as a preventive. It was in his fourth report that he spoke of the use of bile in this way, and his method is best explained in his own words, which are: "One is able to render immune healthy cattle with the bile of such that have succumbed to rinderpest. In this case one hypodermic injection of 10 cc. is sufficient. This immunity sets in on the tenth day at latest, and is of such an extent that four weeks afterwards 40 cc. of rinderpest blood could be injected without any injurious result. I therefore conclude that the immunity produced in such manner is of an 'active' nature."

It will be seen from these statements that although Koch reported that bile did not contain the contagium of rinderpest, it was yet able to produce active immunity. As a matter of fact, it was later conclusively proved by Kolle[2] that rinderpest bile does contain the virus of the disease. The same observer also stated that bile of the third or fourth day may produce rinderpest.

It was found by Koch that only the bile from animals that had been ill for six days could be used for protective work. It may be accepted as a fact that all rinderpest biles do contain the virus, but that after the fifth day of the disease it is modified by the formation of some kind of protective substance formed within the bile.

Lingard[3] says the passive immunity conferred by the injection of rinderpest bile is brought about by the action of the contained nucleo-proteid and lecithin, and the degree and duration of the immunity bear a direct ratio to the quantities of these bodies in the injected bile. He says that "the contagium of rinderpest only effects an entrance into the bile in the gall bladder on the fifth or sixth day of the disease," and "when a sixth day bile is utilised, with the *materies morbi* of rinderpest present, the contagium is more or less kept in check by the action of the nucleo-proteid present, and produces in the injected animal a very modified form of the disease, but nevertheless the result is a transitory *active* immunity, which may persist for a period of three to five months, according to the degree of virulence possessed by the contagium in the injected bile." The same observer claims to have conferred a passive immunity on plains cattle and buffaloes by successive doses of normal bovine bile containing nucleo-proteid and lecithin in sufficient quantities.

Koch used the biles of animals that had been inoculated with rinderpest and were killed on the sixth day of the disease. He stipulated that only biles with certain characteristics should be used.

They had to be clear, of a dark green colour, sweet smelling, and to give a white froth on being shaken up. They had to be collected asceptically in sterile flasks. Bile of this description could not be kept long, even in ice. In South Africa, to obtain bile answering to this description, 3 to 7 per cent. of herds were sacrificed. Rogers[4] found in India that only 17 per cent. of biles from animals that died from rinderpest were fit for use by this method. Later, Turner and Kolle[5] said that all biles not foul or bloody were safe, and these authorities recommended the mixing together of biles, and found that the great majority of biles obtained from animals slaughtered on the sixth day of the disease were fit for use by Koch's method. Animals that die rarely supply suitable bile for Koch's method. Hence, in order to obtain a large quantity of suitable bile slaughter is necessary. It is no doubt advisable, as Turner and Kolle[5] recommended, to mix all apparently suitable biles together before use.

Although in India it was not found that biles of six days or upwards ever produced the disease, there was a good deal of rather conclusive evidence that in many cases in South Africa the disease was produced by bile inoculation. It is very unlikely that biles of suitable age, even if blood-stained, do so, because Koch himself and others found that bile and virulent blood, in equal proportions even, could be injected without producing disease.

It is possible that, in those cases not due to natural infection, young biles, i.e., taken before the sixth day of the disease, were inadvertently used.

The injection of the bile is usually made in the dewlap, and a local swelling the size of a cricket ball results. The swelling is hard and painful, but gradually subsides. No systemic disturbances are usually seen. A local abscess may result if the bile is in a state of decomposition, but this does not seem to be detrimental to the process of immunisation. Larger doses than 10 cc. do not appear to increase the immunity conferred. An injection of bile does not surely produce immunity until ten days have elapsed since its injection, though immunity may in some cases be produced on the sixth day. Animals which may have contracted the disease before the injection, and which may be exposed to infection before ten days have elapsed, may die from rinderpest.

The duration of the immunity conferred in successful inoculations in South Africa was put down to average about four months. Occasionally it was only three weeks, but even in a single herd inoculated with the same bile the respective periods of immunity were found to vary. Whether preventive inoculation according to Koch's bile method produces an active or a passive immunity must be left an open question. The important point is that the immunity conferred is not a lasting one, and in some cases it is of very fleeting duration.

Results.—No doubt excellent results from this method were obtained in certain cases in South Africa. Turner and Kolle[5] reported that in Basutoland 70,000 out of 100,000 animals were saved, of which 90 per cent. would have perished, if the disease had not been checked. However, it appears to have been practically discarded for the simultaneous method of Turner and Kolle, which is discussed later.

Nicolle and Adil Bey,[6] working in Turkey, did not care to use bile if serum were available. The method does not appear to have gathered

adherents in Russia either. A number of experiments were made by this method by Rogers[4] at Muktesar Laboratory in India. Both plains and hill cattle were experimented on. The former class are most unsatisfactory as experimental animals, owing to a large percentage being partially or wholly naturally immune. With regard to plains animals, it was found that those immunised by Koch's method were afforded an immunity of between three-and-a-half and five months—probable average four months.

These results agreed very closely with those obtained in South Africa, Russia, and Turkey. Raymond[7] (Bengal) claimed to have produced immunity of a year's duration with bile taken from buffalo calves which had been inoculated with rinderpest, but the results appear to have been by no means conclusive.

With regard to the hill animals used by Rogers[4] in bile experiments by Koch's method, it was found that little or no immunity was conferred. Animals tested at periods of less than four and three months, respectively, died of virulent rinderpest, and it was even found in one case that no immunity was existent on the tenth day after the bile inoculation.

It should be mentioned that the biles taken for Rogers' experiments were taken immediately after death. Death usually occurred in the experimental animals on the seventh or eighth day, and biles of this age were found as satisfactory as those which happened to be obtained on the sixth day.

Most of Rogers' experiments were, however, done with biles from animals that died on the sixth day. In some of his experiments the bile was used on the same day as obtained, whilst other biles were kept from three to five days in the cold room of the Laboratory. Again no difference was observed in the results obtained. Rogers confirmed the statement that biles taken on the third and fourth day of the disease conferred no immunity in hill cattle.

So far as field work in India is concerned, inoculation by this method was never general. Hagger[8] inoculated 200 animals at the Government Cattle Farm at Hissar; 22 of these animals were attacked between the first and the tenth day, of which two died. He admitted that the disease was dying out at the time of inoculation, and said, moreover, that the bile used was neither clear nor of the stipulated colour. The serum method was preferred by this observer, hence few inoculations with bile were performed.

Raymond inoculated 261 animals belonging to a European planter in Bengal with buffalo bile with successful results. He found that in the large majority of cases immunity was conferred for an average period of five months. Later he[7] found that some animals were still immune at the end of a year.

No other field work with the bile method in India appears to have been practised. There were many objections to it, as explained later, and other methods were preferred.

Advantages of Koch's Bile Method.—No systemic disturbance of importance in normal cases results, and, consequently, there is no loss of milk in cows, or fear of abortion. There is little fear of spreading rinderpest or other diseases by this method when operating on clean herds. This assertion is based on the assumption that bile suitable for injection purposes does not convey rinderpest. One inoculation

only is required, whereas in some of the other methods two or more are necessary.

Disadvantages of the Method. — The disadvantages appear to outweigh the advantages under any conditions, but in India the difficulties associated with this method are well nigh insurmountable. If the immunity conferred had proved to be a lengthy one, no doubt strenuous efforts would have been made to overcome the difficulties, because in one respect the method is peculiarly applicable to that country. That is, that blood is not brought into use at the time of inoculation. A great disadvantage is that immunity does not become absolute until ten days have elapsed after injection. This renders the inoculation of contact animals in diseased herds a delicate proceeding. Some animals would be sure to become diseased from natural infection within that period, and the ordinary Indian villager would put the worst possible construction on this. No doubt the method might have proved useful in healthy villages, but this plan would rarely be adopted, as the native believe in " leaving well alone," and if accidents occurred in this case the remedy would be much worse than the disease. The difficulty in obtaining bile is very real in India. For Koch's method bile must be fresh, and therefore it is practically necessary to obtain it on the spot. Slaughtering cattle in villages is out of the question. This difficulty might be got over by slaughtering cattle, or preferably buffaloes at certain depots, and no doubt the question of the preservation of the bile could be managed for at least a short period. A certain amount of bile can generally be obtained at the scene of an outbreak from dead cattle, but an immediate *post-mortem* when death takes place is indicated, and this might not always be convenient. In this case, also, it would be a difficult matter to judge how long the animal had been ill, and in virulent cases biles of the fourth and fifth day might come to be used. As a matter of fact, it is possible to take bile from the living animal fairly easily.

It was found by Rogers[4] that from seventy-six cattle that died of rinderpest on the sixth, seventh, or eighth day only twenty biles were suitable for use by Koch's method. The average amount of bile collected from these twenty cattle was 190 cc. A simple calculation will show that seventy-six hill cattle would therefore only supply sufficient bile for 380, or 20 per cent. Plains cattle would no doubt furnish much more, as they are larger, but so many would be found to be immune from rinderpest that much disappointment would arise. This difficulty could be got over to some extent by using buffaloes, but these animals are much more expensive and a large percentage are immune. To sum up, Koch's bile method in India is practically only suitable for cattle kept under European management, and it is even then by no means ideal.

II.—GLYCERINE BILE METHOD OF EDINGTON.

It was found by Koch[1] that glycerine destroyed the virulence of rinderpest blood. Edington,[10] therefore, took advantage of this fact to mix glycerine with bile in order to destroy the virus of rinderpest in that fluid. The belief that bile was capable of frequently causing fatal attacks of rinderpest, referred to above, was sufficient reason to

justify the employment of a modified bile incapable of spreading the disease. Although the immunising properties of this mixture were greatly modified, yet Edington found that glycerinated bile in doses of 15 cc. gave sufficient protection to allow of 0·1 cc. virulent blood being inoculated ten days afterwards.

Glycerinated bile (Edington) is prepared by mixing two parts of bile with one part of glycerine, and the mixture has to be kept for eight days before being used. It was also advised that, in addition to the blood inoculation on the tenth day, a further dose of 1 cc. of virulent blood should be inoculated to make the immunity conferred stronger still. Many herds in South Africa are said to have been protected by injecting glycerinated bile in doses from 15 to 20 cc., according to the size of the animal, followed ten days after by a small dose of virulent blood.

The immunity conferred is dependent upon a reaction occurring from the dose of virulent blood, but an important point is that if a too strong immunity is conferred by the glycerinated bile no reaction occurs. In the latter case immunity is very short and bad results may accrue.

It was reported that 393,777 animals were inoculated in South Africa with a loss of only 8·25 per cent. in twenty-three districts in which it was extensively used. Edington [10] claimed that by this method more biles could be used than by Koch's method ; but, with regard to the latter, later observers showed that more kinds of biles could be used than Koch originally allowed. Turner and Kolle [5] admitted that glycerinated bile possessed feeble immunising powers owing to certain chemical substances contained in it. The bile is, however, deprived of all active immunising power, as the glycerine kills the virus contained in the gall.

Results.—In India Rogers [4] inoculated a series of twelve plains cattle by this method, and in only four did any sign of rinderpest follow an inoculation of virulent blood ten days afterwards. No severe symptoms occurred in any of these animals. It may be allowed that they could claim to have attained a very strong immunity. Eight animals did not react, and consequently they were probably only protected for a short period. It is possible that in these cases some of the animals were naturally immune, a difficulty commonly met with in plains cattle.

Out of twelve hill animals inoculated by Rogers [4] with glycerinated bile, nine died after the blood inoculation, and two were very ill. He therefore "regretfully admitted that the glycerine bile method not only fails to confer immunity on Kumaon hill cattle, but actually is fatal to them as frequently as is inoculated rinderpest."

No inoculations of any account were performed in the field in India by this method. Rogers [4] mentions that Walker did three animals at Bareilly with satisfactory results.

Advantages of glycerinated bile.—Biles can be kept for a long period ready at hand ; preserved in this way, it is claimed that they retain their properties for upwards of a year.

If a definite reaction is obtained after the blood inoculation an active immunity of long duration is conferred. A larger percentage of biles can be used than in Koch's method.

Disadvantages of glycerinated bile.—As in Koch's method, immunity

is not conferred for ten days after the inoculation has been performed.
Many animals fail to react after the blood inoculation, and, conse-
quently, immunity of short duration is conferred. The period is
probably much less than with Koch's method. On the other hand,
sometimes very severe reactions occur, a state of things which Indian
cattle owners would not appreciate.

Much time is lost, viz., eight days, before the bile is ready for use.
It was found in India that the method was dangerous in hill animals.

A double inoculation is necessary, which makes the method
cumbersome, and certainly distasteful to cattle owners. An inocula-
tion of blood is required, which entails the risk of spreading not only
rinderpest but other diseases. To a great extent the same objections
with regard to obtaining the bile in the method of Koch also apply
here.

III.—OTHER METHODS ENTAILING THE USE OF ORDINARY OR MODIFIED BILES AND MIXTURES OF BILE AND OTHER FLUIDS.

In order to avoid risk of infection from bile, Rogers[4] passed a *mix-
ture* of biles through a porcelain filter. Other observers in Turkey
and South Africa had previously found that filtered blood was no
longer virulent. Rogers used all biles except those red with blood
or light brown in colour. He inoculated with small doses of virulent
blood after ten days had elapsed from the injection of bile, as in
Edington's method. The results obtained were similar, but somewhat
better than those in the glycerinated-bile method. The method
practically possesses the same advantages and disadvantages as the
latter. The time lost in the process of glycerinating the bile is saved,
however. Biles sterilised by heat can be utilised in the same way, and
they can be kept for a long time by adding half per cent carbolic acid.

The brothers Krause[13] contended that simple rinderpest bile, as a
preventive method, was superior to any other. They were convinced
that it was quite capable of producing the disease, and that when it
did so the period of incubation was eight days. If the immunising
substances in the bile were sufficiently powerful no outbreak of disease
would occur. They did not recommend the use of bile in herds
already attacked with the disease. They advocated the use of serum
as a curative if rinderpest appeared among cattle inoculated with bile.

They used all biles not red or foul, and made two inoculations.
The first inoculation consisted of 8 cc. of bile three to seven days old,
and twelve to fourteen days after a second inoculation of 15-20 cc.
of bile twenty-four hours to five days old had to be given. Ten days
after the last bile inoculation it was advised that 5 cc. of virulent blood
be inoculated. They claimed for this method that it conferred an
immunity of two months duration by the bile inoculations, and a
longer period if 5 cc. of virulent was used. The method was compli-
cated, and it seems never to have been used in the field to any appreci-
able extent.

The brothers Krause also considered that it was possible to prepare
an artificial rinderpest bile.

Their formula was :—To an immunised serum add 5 per cent nor-
mal bile salts, or healthy bile and mucin. Add 10 per cent virulent
rinderpest blood, and allow to stand for twenty-four hours.

Koch[1] made several experiments by mixing larger or smaller quantities of virulent rinderpest blood with rinderpest bile, and found that bile was able to make a considerable quantity of rinderpest blood innocuous. He obtained immunity after an injection of 5 cc. bile and 5 cc. virulent blood. It was thought that if bile were mixed with virulent blood it would be strengthened as a protective agent. This supposition does not seem to have been borne out by experiment.

Rogers[4] performed a number of experiments on Indian cattle with mixtures of bile, blood, and fortified serum. His main idea was, if possible, to manufacture an artificial bile. In the first place, he found that in order to neutralise rinderpest blood, if injected into susceptible animals, about ten times as much serum as blood was necessary.

He then found that 1 cc. of a bile solution (made by dissolving the ox gall of the British Pharmacopœia in sterile water until the specific gravity was 1020.), neutralised 10 cc. of virulent blood. Varying mixtures of serum, blood, and normal bile were then used, but no success in conferring immunity was obtained. The same observer also tried mixing filtered bile and virulent blood. The experiments were not successful. In one animal a mixture of 20 cc. of virulent blood and 5 cc. of filtered bile did not cause the disease, yet the animal died on being inoculated with rinderpest blood ten days afterwards. Unsuccessful experiments were also made with mixtures of ordinary bile, filtered liver extract from an animal dead of rinderpest, and virulent blood.

If one sums up the different methods necessitating the injection of bile, modifications of bile, and mixtures of bile and other fluids, the following deductions seem indicated. Biles that answer to the characters prescribed by Koch in his method, and perhaps other biles to a less degree, contain substances, probably of an albuminoid nature, which inhibit the virus contained in such biles.

After inoculation, absorption of the virus, modified in some peculiar way, takes place, and immunity is conferred. In certain cases it is to be presumed that the virus enters the system in an unmodified form, and disease results. The peculiar modification of the virus in successful inoculations is such that no symptoms of the disease appear, and a passive immunity of rather long duration is produced. Lingard[3] prefers to call this a transitory active immunity.

Artificial modifications of bile may render the operation of inoculation safe, in the sense that the disease itself is never conferred by them. They have, however, little protective influence as compared with Koch's unmodified bile. Lingard's experiments with normal bile appear to have been confined to plains cattle and buffaloes, many of which are found to be wholly, or partially immune.

With regard to mixtures of bile and other fluids, with the idea of preparing an artificial bile, no remarks seem necessary, as the experiments so far have failed.

A very striking point brought out, however, is the very large relative quantity of virulent blood that can be neutralised by mixing it with bile. It took ten times as much serum to neutralise virulent blood, but one part of normal bile neutralised ten parts of virulent blood (Rogers). No immunity was conferred, which points to the virus having been destroyed *in situ*. It may be mentioned that in these experiments large swellings were produced where the mixtures were injected. The local inflammation probably had a good deal to do with

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the destruction of the virus. It is possible that the local swelling induced in Koch's method has more to do with the results attained than appears at first sight.

IV.—THE SERO-THERAPEUTICS OF RINDERPEST.

Semmer, in 1895, working in Russia, ascertained that the blood of an animal that had recovered from rinderpest possessed passive immunising qualities if injected into a healthy animal.

Pitchford and Theiler in Natal, and later Danysz and Bordet in the Transvaal, and Spreull and Campbell in Cape Colony, used the defibrinated blood of animals recovered from the disease to confer protection, with a considerable measure of success. The immunity conferred was, of course, only of a passive nature, but in many cases it was turned into an active one by infecting such protected animals with the disease by mixing them with diseased cattle, or by smearing the discharges of such animals on their noses.

At first, in South Africa, defibrinated blood collected on the spot was used, but later serum depôts were formed and serum supplied.

It is obvious that defibrinated blood collected in the field possessed many disadvantages, especially as it appears to have sometimes been obtained in a very crude manner. Defibrinated blood is weaker than serum in the relation of 30 cc. to 20 cc. (Turner and Kolle[5]). Koch[1] (fourth report) used the serum of salted animals in doses of 100 cc. to protect against experimentally inoculated rinderpest. He obtained the serum by the clotting method. In order to keep it, he recommended drying it in a vacuum apparatus. Defibrinated blood had to be used in doses of 100-200 cc., and its strength was very uncertain. Koch and others consequently preferred bile.

It was not until Turner and Kolle[5] perfected a method of preparing a fortified or hyper-immunised serum that methods involving the use of serum became popular in South Africa. These observers claimed to manufacture a serum of such a strength that 20 cc. was efficacious as a cure in the early stages of the disease, and 50-100 cc. in the later. It had the additional advantage that it could be kept without losing its qualities. It was prepared by giving to a salted animal increasing doses of virulent blood up to 1000 cc. Successive doses of 100, 200, 500, and 1000 cc. were injected subcutaneously. The larger dose in each case was given after the reaction from the smaller one had subsided.

After the last injection the animal was bled once a week, for three successive weeks, and the fortifying process again resorted to in larger doses. Half per cent. carbolic acid was added to keep it.

By the method of Turner and Kolle some time is lost (nearly two months) in fortifying an animal. Nicolle and Adil Bey[6] used what they termed the "brutal" method instead, and prepared fortified serum in fifteen days. They gave 4 litres of virulent blood and 25 cc. of serum by the simultaneous method (*see* later), and bled fifteen days after, *i.e.*, when the reaction had subsided. They bled animals fortified in this way every week for six weeks, and found that serum at the sixth bleeding was as strong as that of the first. Doses of 25 cc. were found to protect against a dose of virulent blood given a few days later, or from natural infection. Later they used as much as 8

litres of virulent blood at one injection to fortify, and found the serum still more active.

In India, serum was first prepared by Rogers[4] according to the directions of Turner and Kolle. The "brutal" or rapid method is now usually employed, using both cattle and buffaloes. The blood is defibrinated in the flask and then centrifugalised. The serum is then carbolised and placed with other anti-rinderpest serum in bulk. When a quantity has been collected it is tested and bottled off. Serum-making animals have their temperatures taken night and morning, and unless they are perfectly healthy their blood is not employed. Lingard[15] advised that animals under three years of age should not be used, as "they yield a serum the protective power of which may be five to eight times less than that yielded by the same breed of animal above three years of age." The same observer also said that : "By the rapid method large doses of virulent blood injected into the serum animal do not yield proportionate results in producing a high protective serum. For, it has been found that, if doses over 6000 cc. to 7000 cc. are injected at one time, a weak or attenuated form of serum is the result."

Nicolle and Adil Bey[6] considered that the cattle of the steppes, being less susceptible to rinderpest, were most suitable for serum-making, as they would produce a stronger serum. This was not found to be the case in India, where the hill animals produced the strongest serum.

Lingard[15] found that sera rapidly lost their qualities when exposed to the high temperatures registered in the plains of India during the hot season. At Muktesar laboratory, however, at an altitude of 7600 ft., the sera were found to retain their protective qualities for long periods. In preparing serum care should be taken to select good, strong, healthy animals, and the aim in view is to prepare as strong a serum as possible. Large doses of virulent blood should be injected with the object of obtaining good temperature reactions. Larger doses of virulent blood than are absolutely necessary are waste of material. It is important to see that the blood used is free from the virus of any other disease than rinderpest. It should be taken at the height of the fever, before diarrhœa commences. It should be defibrinated, strained through muslin, and injected as soon as possible, with due aseptic precautions. It is well not to inject too much blood in one place, or absorption is delayed and abscesses may result. The curative effects of even very strong serum are not very great. When once diarrhœa has set in no amount of serum appears to exert any restraining influence on the course of the disease. On the first or second day of the fever, before other symptoms have set in, fortified serum in doses of 100 to 200 cc. sometimes checks the course of the disease.

The Use of Serum Alone as a Preventive.

As mentioned above, both defibrinated blood and serum were used in South Africa by several workers. Danysz and Bordet are said to have salted many animals in the Transvaal with serum alone. In South Africa it was given up almost entirely in favour of the simultaneous method of Turner and Kolle (referred to later). In Turkey

Nicolle and Adil Bey,[6] and Nencki,[4] in Russia, used fortified serum alone, and appear to have preferred it to any other method. The immunity conferred was said to last for several months, and the herds were at any rate protected through the particular outbreak to which they were exposed. The conditions of rinderpest in Russia closely resemble those in India, and, as it was considered that serum alone was an effectual method in that country, it was likely to suit Indian conditions also. With regard to the dose of serum required to protect, that must depend upon the strength of the serum, the size and breed of the animal, and the period that one wishes to protect for. In India the strength of the serum is tested according to the simultaneous method of Turner and Kolle. By this means it is found what quantity of serum will protect an animal from a severe attack of the disease, the disease having been given at the same time as the serum. The susceptibility of the different breeds varies so much in India that doses vary accordingly. Eighteen times more serum was found to be required for hill animals than for ordinary plains animals, and the dose is usually fixed in that ratio. With regard to the susceptibility of other breeds in different parts of the country, experiments are still being made to enable some sort of scale to be prepared. The serum is usually sent out at so much per animal of 600 lbs. as was the custom in South Africa. Fairly accurate estimates of the weight can be made by measurement, in the absence of weigh-bridges. It is not practicable, of course, to weigh or measure more than a few animals, nor is it necessary. The inoculator soon learns to give a fairly accurate guess at the weight of an animal at sight, and for the serum alone method great accuracy is not required. The more serum that can be given the better, within limits, but serum is rather a valuable commodity, and has to be used with discretion. In the serum alone method it is usual to give rather more than the dose specified as correct for the simultaneous operation, and a minimum of 5 cc. is the rule.

Working in India, on no account should sick animals be inoculated, as that might tend to destroy the confidence of natives. All contact animals that have not recovered, or that are not ill, should be inoculated and then all mixed together. Some animals may thus become sick and acquire an active immunity. Disinfection of the cattle sheds and surroundings is essential, or in course of time the inoculated animals may be re-infected. In India the hot sun is a great factor in this respect.

Results of Serum Alone in India.—As in other methods, experiments were first made in the laboratory, and then tried on cattle belonging to Government or public bodies before inoculations were applied generally. In the field Walker[4] in 1899 inoculated thirty-one cows and thirty-eight milch buffaloes at the Bareilly Lunatic Asylum Dairy. This method was used instead of the simultaneous because it was desirable that the milk supply of the dairy should not be interfered with. No cases occurred after the inoculations, and the milk, which was measured, did not fall off in any appreciable degree. During 1900 Hagger[16] inoculated over 1000 animals by this method with successful results. Since then the method has become fairly general in India. Between April 1902 and April 1903[19] 24,457 animals were inoculated in actual outbreaks in the Punjab, out of

which only 2 died after inoculation. Villages in which inoculations took place were visited regularly for three months to watch results. In the same year 11,472[17] animals were inoculated in Bengal, and the total for the whole of India and Burma came to 42,871, with 139 deaths. The uninoculated animals which died during the course of the outbreaks (as far as could be ascertained) came to 1074. These results speak for themselves, and although, no doubt, the method is far from ideal, it has been of great service in India, as it is peculiarly adapted to the conditions met with in that country.

Advantages of Serum Alone.—It affords immediate full immunity, and it may cure disease in the incubative period.

The material used can be stored and kept ready for use. It can be transported in bottles and used as *medicine*. This is an important point in India, where medicine is rather popular than otherwise. The patent use of body fluids would not be regarded with favour. The simplicity of the method is a great advantage, as most of the inoculations have to be done by the native staff. There is very little fear of disease being conveyed, or any septic infection being set up, local or otherwise. No great accuracy in determining the dose is required so long as a sufficiently large one is given.

One inoculation only is necessary. Animals can be worked or milked just as usual.

There is a practical certainty of getting good results (within a limited period) if the inoculations are carried out properly.

The cattle owners are put to little or no trouble themselves in the matter, as they would be if any segregation were necessary, or the animals became ill. The method inspires immediate confidence, being so harmless.

Disadvantages of Serum Alone.—These are practically summed up when one has stated that the immunity conferred is of rather a short duration. In most cases, however, if sufficient doses of serum are given, the immunity conferred is long enough to carry over the particular outbreak. Occasionally, if the disease is particularly virulent, or the serum particularly weak or scanty, unfortunate results may occur.

Serum Simultaneous Method.

This method was instituted by Turner and Kolle,[5] and consists in injecting simultaneously a certain dose of serum on one side of the animal (usually behind the shoulder) and a dose of virulent blood on the other side, or in another part of the body. The object in view is to pass the animal through a slight attack of the disease, and thus confer an active immunity. The dose of serum has to be determined with some accuracy to bring about this result. If the amount injected be too small the animal is likely to get a severe, or even fatal, attack. On the other hand, if it be too large no reaction occurs, and simply a passive immunity results.

In South Africa the dose of serum could be determined with comparative ease, as all the animals were practically equally susceptible to the inoculated virus. The serum used was fortified serum, as first prepared by Turner and Kolle. It was tested at the depôt, and at the time of injection the quantity to be given was regulated according to the weight of the animal. The weight had to

be determined by measurement. If a too severe reaction occurred more serum could be injected for its curative effect.

For this method fresh virulent blood is necessary. It can usually be obtained on the spot, but in India this is often rather a difficult proceeding, requiring great tact. Virulent blood should be drawn from an animal with a high temperature and suffering from rinderpest. It should not be taken if diarrhœa has commenced. It is important to be quite sure the disease is rinderpest, and the animal is not suffering from tick fever or any other disease as well. Rinderpest blood rapidly loses its activity—it cannot certainly be kept virulent more than three to four days at summer temperatures, or more than six to eight days in ice, and a high temperature or dessication destroys the virus rapidly.

Defibrinated or citrated blood ($\frac{1}{1000}$) in volume of 200 cc. remains virulent during twelve days at 15° to 18° C. (Nicolle and Adil Bey.[6]). Blood can be conveniently carried about in a live sheep inoculated with rinderpest. Turner and Kolle claimed that 90 per cent. of the animals inoculated in South Africa reacted. It is said that many thousands were treated, particularly in Rhodesia, with a loss of only 1-2 per cent.

Results of the Serum Simultaneous Method in India.—The laboratory experiments performed by Rogers[4] at Muktesar proved that animals that reacted attained a very high degree of immunity. It was found that even if the reaction was purely a thermal one, and no other symptoms occurred, a very strong immunity was conferred. This was useful information, as it was not desirable to produce severe symptoms, on account of the ignorance of the cattle owners. In South Africa as severe an attack as possible consistent with safety was the object. In order to test serum, repeated inoculations were, and are, made with cattle at Muktesar by the simultaneous method. Many of the cattle are tested with virulent blood at long intervals afterwards, to see if they are still immune.

Although some of the plains animals inoculated by Rogers reacted, yet a great many failed to do so with relatively similar doses. The weights of the animals were accurately determined on the weighing machine. These uneven results were put down to the variation in susceptibility in these cattle.

Most irregular results were got with hill animals also by this observer, and he reported that the method was not reliable in this particular breed of animal. The reason was probably that at that time the enormous difference in susceptibility between these breeds of cattle had not been recognised. It was found later that fairly safe reactions could usually be obtained in hill cattle if sufficient serum was used. Lingard considers eighteen times the dose required for plains cattle necessary for hill cattle. Luckily the latter are generally small, but even then they often require a larger absolute dose than plains animals.

In 1899 Rogers, Hagger, Holmes, and Walker performed some inoculations in the field by this method, with, in most cases, very satisfactory results. In other cases reactions did not occur, or they were not detected. These inoculations were, of course, performed with great care, and under the direct superintendence of the officers themselves, as at that time the process was regarded as experimental.

Hagger,[16] in 1900, however, had bad results in the Punjab, 24 animals dying out of 128 inoculated by one of his native assistants. This observer condemned the method for India as unsuitable, and recommended serum alone as more likely to give good results. The method is now rarely used in India, though there are occasions when it can be usefully employed. This is especially the case when one inoculates cattle belonging to persons who are capable of understanding its dangers, and are content to run some risk in order to obtain a distinct advantage.

In many ways the method is ideal, but, to be practised with success, a uniform scale of susceptibility in the cattle and a certain degree of intelligence in their owners are necessary.

Advantages of the Serum Simultaneous Method.—Immediate immunity is conferred. In successful inoculations—*i.e.*, when good reactions occur without dangerous symptoms arising—the immunity conferred is active, and probably persists in most cases for the animal's life. Even when reactions do not occur the inoculation has not been without service, though it may be disappointing. Practically only one inoculation is required. The serum can be kept ready for use, though the blood is sometimes a difficulty.

Disadvantages of the Serum Simultaneous Method.—There is a certain amount of risk in the method, especially in countries where the cattle vary in susceptibility. It requires a good deal of skill and care to bring about good results in such countries. Consequently, in India only a selection from the native veterinary staff could be safely trusted with it on their own account. As a matter of fact, in that country, before inoculating any number of animals in a certain locality by this method, the serum should be tested on a few animals on the spot, to arrive at the correct dose. Even then the operator might be disappointed by irregular results, and valuable time would have been lost.

That virulent blood has to be used is rather a serious disadvantage in India. In the first place, it has to be obtained. In some cases this difficulty is only surmounted by taking the blood with one, and then it may not be fresh.

It is obvious to the most casual observer that blood is being used, and the ordinary Indian agriculturalist has scruples in this respect. They can usually be got over by judicious argument, but if any deaths do take place it is quite apparent to the owner that the inoculator caused it by injecting blood. It is possible to spread the disease in this method through the medium of the reacting animals.

Other Methods Entailing the Use of Serum.

Hutcheon[12] regarded the serum simultaneous method of Turner and Kolle as ideally perfect, but found it was difficult to invariably ensure the requisite reactions to justify that character. He therefore adopted the method of giving the virulent blood forty-eight hours after the serum, in the hope of ensuring more constant reactions. In many cases a second injection of serum was found necessary in this method, which made it complicated.

Rogers[4] recommended a reinoculation of virulent blood in animals that did not react in the simultaneous method. The blood has to be given ten days after the double inoculation of blood and serum.

He used rather large doses of serum in the first inoculation to guard against mortality, and claimed that all animals that did not react at first would when reinoculated.

This method is also complicated, necessitating careful watch being made for reacting animals, and in many cases a second inoculation.

Koch [1] (fourth report) was in favour of a mixture of serum and virulent blood. He considered an inoculation of this mixture would confer active immunity, and used 99 parts of serum and 1 cc. of inoculated blood, injecting 20 cc. of the mixture. He claimed to obtain a certain basis of immunity, which he strengthened by means of repeated blood inoculations. Rogers [4] found that mixtures of serum and virulent blood failed to confer immunity.

V.—EXPERIMENTS WITH MODIFIED BLOOD.

Edington found that citrated blood, kept for such a time as to ensure the death of the contagium, in doses of 20 cc. conferred a certain degree of immunity.

Koch [1] tried mixing virulent blood in varying concentrations with glycerine, phenol, distilled water, and normal salt solution, respectively, but no modified virus capable of being used for producing immunity was elaborated.

Rogers [4] tried dried blood, sterilised blood, oxygenated blood, and hydrogenated blood, with similar results. The latter also tried blood from animals when reacting, and after reacting, to virulent blood in the process of serum-making. He considered that the disease was produced, slightly intensified, when the blood was taken during the temperature reaction. When the temperature was declining after injections of virulent blood into salted animals, the blood might, or might not, produce the disease. If it did produce it, it was in a severe and unmodified form. When no reaction occurred the disease was not produced in susceptible animals, and no immunity was conferred. Blood passed through a porcelain filter has been tried as a preventive by the writer and others with no success.

The above enumeration of the experiments conducted by different persons with blood in attempting to modify the virus for a useful purpose are interesting, but, as no practical results were obtained, they are not worth discussing.

VI.—PASSAGE THROUGH ANIMALS.

It may be said that ruminants only are susceptible to rinderpest. Nicolle and Adil Bey [6] injected 5 cc. of virulent blood into the peritoneal cavities of rabbits and pigeons. From these animals they inoculated cattle on the fifth day. The result was—no disease and no immunity. Koch [1] carried rinderpest through goats and sheep to the seventh generation. He thought that the rinderpest virus after repeated passage through goats became slowly attenuated, but was inclined to believe that as far as sheep were concerned it became more virulent. Nicolle and Adil Bey [6] did not think the virulence was augmented when passed through sheep, and that irregular results were obtained after passage through goats. The susceptibility of goats and sheep probably varies considerably. In India sheep become ill but rarely die. Passage through sheep did not appear to

modify or intensify the virulence of the disease to any appreciable degree. Goats generally die, but the disease in them runs rather a long course. They die of fever and progressive emaciation in from nine to sixty days. Females always abort. Passage through goats in India did not appear to alter the virulence of the virus.

Conclusion.

In conclusion it may be said that there is yet room for a safe and effectual method of inoculation against rinderpest. So far as India is concerned the serum alone method is doing a good deal of good, and is filling a gap. The expense incurred in preparing and using the serum is considerable, but it is probably more than covered by the great saving of cattle that without it would doubtless die of rinderpest. Cattle in India are almost the most important asset to agriculturalists, and the country practically exists by agriculture. It is hoped that a really efficient and economical method of preventive inoculation for rinderpest suitable to that country will eventually be evolved.

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IMMUNISATION AGAINST HORSE–SICKNESS BY THE METHOD RECOMMENDED BY PROFESSOR KOCH.

By CHARLES E. GRAY, M.R.C.V.S., Chief Veterinary Officer, Salisbury, Rhodesia.

THE following is a tabular record of the results of the experiments in horse-sickness initiated by Dr. Koch and brought to a termination by this Department after his departure from Bulawayo.

The results which have been obtained confirm the unanimous opinion of previous investigators as to the marked difference in the susceptibility of individual horses, and seem to suggest that, although the artificial establishment of an active immunity against horse-sickness in susceptible animals, by gradually increased doses of virulent blood, alternated in the early stages of treatment with the injection of serum prepared from the blood of highly fortified salted horses, may be possible without any reaction following such treatment, still, unless the process is extended over a very long period, and unless the doses of virulent blood are increased much more gradually than is recommended by Dr. Koch, sooner or later a stage is reached at which the natural resistance of the animal is overcome by the increase in the amount of virulent blood injected, and an attack of horse-sickness supervenes, the severity of which is not materially modified by the previous injection of smaller quantities of virus.

Unless a distinct reaction follows inoculation on the lines at present laid down, I incline to the belief that no real or lasting immunity is established, even if the animal inoculated has withstood inoculation with a dose of 5 cc. of virus which has produced a fatal reaction in other horses subjected to the same treatment.

The results in the case of horses 32 and 40 appear to indicate that this may be the case. Horse 32 (Case III.) was regularly immunised without any distinct reaction occurring. The final dose to which he was subjected was 5 cc. of preserved defibrinated blood—a dose which was followed by a fatal attack of horse-sickness in other cases, but his resistance to this amount of virus did not prevent him from developing six weeks later what his owner considered to be an unmistakable attack of horse-sickness, characterised by the usual supra-orbital swelling, dulness, and a disinclination to food which lasted for three days. Horse 41 (Case VI.), which also resisted inoculation with 5 cc. of the same sample of blood without reaction, succumbed soon afterwards to a dose of 5 cc. of another and more powerful virus after a somewhat prolonged period of incubation.

Of the twenty horses whose treatment was begun by Dr. Koch and finished by the Department, twelve reacted and died of horse-sickness. The numbers of these animals are 29, 36, 38, 41, 43, 47, 48, 51, 54, 55, 56 and 61. These numbers include those of seven animals in which the interval between two injections was accidentally delayed without any immediate ill effects resulting therefrom.

Seven animals, including horses 32 and 40, whose temperature record is not given, withstood treatment and will be kept under observation next horse-sickness season.

While these returns seem to show that the lines of treatment laid

down by Dr Koch require important modifications before the process of establishing immunity against horse-sickness can be of any practical use, I must point out that the experiments were carried on through one of the worst horse-sickness seasons we have had for several years ; and, as it is possible that some of the deaths which occurred amongst animals under treatment were the result of natural infection, it might be well to go over the ground again before expressing any positive opinion.

Case I. (*Mule No.* 3).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	1 cc.	—
5th	—	300 cc.
22nd	0·1 cc.	—
26th	—	100 cc.
40th	0·2 cc.	—
44th	—	50 cc.
54th	0·5 cc.	—
58th	—	30 cc.
68th	0·5 cc.	—
81st	1 cc. (stronger virus)	—
93rd	2 cc.	—
117th	5 cc.	—

On the fourth day after the first inoculation with blood the temperature had risen to 102·2°, the highest previous temperature having been 101·4°. On the following day (after the injection of 300 cc. of serum) it was 102°, and on the next day it had returned to the normal (101°). From that time until the ninetieth day the temperature never reached 102°. On the ninety-first and two following days the temperature was 103°, and on the ninety-ninth day it reached 104°, after which it again fell to normal. The highest temperature after the last blood inoculation (5 cc.) was 103·2°.

The experiment was discontinued, as the animal never had a decided reaction, and was therefore believed to have been immune before the first inoculation.

Case II. (*Horse No.* 29).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	—	200 cc.
2nd	5 cc.	—
13th	—	250 cc.
60th	0·25 cc.	—
64th	—	400 cc.
84th	0·2 cc.	—
88th	—	100 cc.
100th	0·3 cc.	—
104th	—	50 cc.
115th	0·5 cc.	—
127th	1 cc.	—
140th	2 cc.	—
163rd	2 cc.	—
176th	5 cc.	—

This animal never reacted until after the last blood inoculation (5 cc). On the fifth day after that the temperature rose to 104·2°, and three days later it reached 106·2°. Death took place from horse-sickness on the eleventh day after the last inoculation.

Post-mortem. — Marked supra-orbital swelling. Amber-coloured effusion into the tissues of the neck. Large amount of liquid in pericardium. Lungs œdematous, and some interlobular emphysema. Mucous membrane of stomach port-wine coloured. Peritonitis affecting the cæcum. Liver congested and enlarged. Urine normal.

Case III. (Horse No. 32).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·25 cc.	—
5th	—	400 cc.
23rd	0·2 cc.	—
27th	—	100 cc.
30th	—	100 cc.
42nd	0·3 cc.	—
46th	—	50 cc.
69th	1 cc.	—
82nd	2 cc.	—
103rd	2 cc.	—
116th	5 cc.	—

This horse reacted slightly after the first and second blood inoculations, but there was no reaction whatever after the final inoculation with 5 cc. The animal was returned to its owner, who reported that a month later it developed a characteristic attack of horse-sickness, from which, however, it recovered.

Case IV. (Horse No. 36).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·1 cc.	—
5th	—	400 cc.
22nd	0·1 cc.	—
27th	—	100 cc.
28th	—	100 cc.
40th	0·2 cc.	—
44th	—	100 cc.
56th	0·3 cc.	—
60th	—	50 cc.
71st	0·5 cc.	—
83rd	1 cc.	—
96th	2 cc.	—
117th	2 cc.	—
130th	2 cc. (stronger virus)	—

After the last inoculation the temperature began to rise on the sixth day, and the animal developed an attack of horse-sickness which proved fatal on the twelfth day.

Post-mortem. — Blood-stained discharge from nostrils. Large amount of fluid in pericardium. Lungs œdematous. Stomach port-wine coloured. Peritonitis with considerable effusion around the stomach. Urine normal.

Case V. (Horse No. 38).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·2 cc.	—
5th	—	200 cc.
16th	0·1 cc.	—
19th	—	50 cc.
32nd	0·2 cc.	—
36th	—	50 cc.
45th	0·5 cc.	—
58th	1 cc.	—
72nd	2 cc. (stronger virus)	—

For some days prior to the seventy-second, on which the last dose of blood was injected (2 cc.), the temperature had been 103°. After the last inoculation the animal developed a fatal attack of horse-sickness, the temperature rising to 105° on the eighth day, and death taking place on the eleventh day.

Post-mortem. — Swelling over the eyes. Marked subcutaneous œdema, and œdema of the diaphragm. Congestion of the stomach. Slight œdema of the lungs, and froth in the trachea.

Case VI. (Horse No. 41).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·2 cc.	100 cc.
16th	0·2 cc.	50 cc.
28th	0·5 cc.	500 cc.
40th	0·5 cc.	—
53rd	1 cc.	—
66th	2 cc.	—
92nd	5 cc.	—
114th	5 cc. (stronger virus)	—

The temperature began to rise on the sixth day after the last inoculation, and reached 105° on the ninth day, death from horse-sickness occurring on the eleventh day.

Post-mortem.—Marked effusion along the trachea in neck and in thorax. Lungs normal, except for hypostatic congestion of the right. No fluid in pericardium. Stomach port-wine coloured.

Case VII. (Horse No. 43).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·2 cc.	—
3rd	—	100 cc.
18th	0·2 cc.	—
20th	—	50 cc.
31st	0·3 cc.	—
35th	—	50 cc.
46th	0·5 cc.	—
58th	1 cc.	—
71st	2 cc.	—
92nd	2 cc.	—
105th	5 cc.	—

After the last inoculation the horse developed a typical attack of horse-sickness, from which it died on the eleventh day. The highest temperature (105·4°) was recorded on the last day.

Post-mortem.—Large amount of froth from both nostrils. Lungs œdematous. Large quantity of liquid in pericardium. Liver enlarged and congested.

Case VIII. (Horse No. 44).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	—
6th	—	200 cc.
17th	0·05 cc.	—
23nd	—	50 cc.
33rd	0·2 cc.	—
50th	1 cc. (stronger virus)	—

This animal died on the fourteenth day after the last blood inoculation, probably as the result of debility. The *post-mortem* showed broncho-pneumonia of the right lung, sllght œdema of the lung tissue, and localised ulceration of the stomach.

Case IX. (Horse No. 45).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	—
6th	—	200 cc.
18th	0·05 cc.	—
23rd	—	50 cc.
34th	0·2 cc.	—
39th	—	50 cc.
50th	0·5 cc.	—
63th	1 cc.	—
76th	2 cc.	—
89th	5 cc.	—

This animal appeared to react after the blood inoculation on the sixty-third day, and between the seventy-sixth and eighty-third days the temperature varied between 102·4° and 106°. The highest subsequent temperature was 102·4°, on the ninety-first day. The animal's immunity to natural infection is to be tested next horse-sickness season.

Case X. (Horse No. 47).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·05 cc.	—
6th	—	200 cc.
18th	0·05 cc.	—
23rd	—	50 cc.
34th	0·2 cc.	—
39th	—	50 cc.
50th	0·5 cc.	—
63rd	1 cc.	—
76th	2 cc.	—

On the eighty-first day the temperature began to rise, and it reached 107° on the eighty-fourth day. Death from horse-sickness took place on the eighty-fifth day.

Post-mortem.—Slight gelatinous exudate along the course of the trachea, and a small quantity of liquid in the pericardium. Lungs congested, some interlobular œdema, and froth in the trachea. Spleen slightly enlarged, and marked congestion of the stomach.

Case XI. (*Horse No.* 48).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·2 cc.	—
5th	—	200 cc.
17th	0·2 cc.	—
21st	—	50 cc.
32nd	0·5 cc.	—
46th	1 cc.	—
59th	2 cc. (stronger virus)	—
72nd	5 cc.	—

The temperature began to rise on the sixty-sixth day, apparently in consequence of the inoculation with 2 cc. of blood seven days previously, and it reached 106° on the seventieth day. Death from horse-sickness took place on the seventy-third day.

Post-mortem.—Distinct supraorbital swelling. Large amount of fluid in pericardium. Lungs congested and froth in trachea. Marked congestion of stomach.

Case XII. (*Horse No.* 50).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st .	0·01 cc.	—
6th	—	100 cc.
17th	0·05 cc.	—
22nd	—	50 cc.
33rd	0·2 cc.	—
38th	—	50 cc.
49th	0·5 cc.	—
62nd	1 cc.	—
75th	2 cc.	—
88th	5 cc.	—

This animal reacted to the blood inoculations on the sixty-second and seventy-fifth days, and also slightly after the last inoculation. Its immunity to natural infection will be tested next season.

Case XIII. (*Horse No.* 51).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	—
6th	—	100 cc.
17th	0·05 cc.	—
22nd	—	50 cc.
33rd	0·2 cc.	—
46th	0·5 cc.	—
59th	1 cc.	—
75th	2 cc.	—
88th	5 cc.	—

The last inoculation provoked an attack of horse-sickness, which terminated fatally on the ninety-ninth day.

Post-mortem.—Froth from nostrils.   Large amount of liquid in the pericardium.   Lungs œdematous.   Stomach distinctly congested. Some peritonitis involving the large intestines.

Case XIV. (Horse No. 52).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	—
6th	—	50 cc.
27th	0·5 cc.	—
39th	2 cc. (stronger virus)	—
52nd	5 cc.	—

This animal had a distinct reaction, which began on the eleventh day, the temperature reaching 106° on the fourteenth day.   It survived the treatment, and will be tested as to its immunity to natural infection next season.

Case XV. (Horse No. 54).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	100 cc.
14th	0·1 cc.	100 cc.
27th	0·2 cc.	50 cc.
40th	0·5 cc.	—
53rd	1 cc.	—
66th	2 cc.	—
79th	5 cc.	—

The horse reacted distinctly after the blood inoculation on the sixty-sixth day, and the last inoculation was followed by an attack of horse-sickness, which proved fatal on the ninety-first day.

Post-mortem.—Froth from nostrils.   Slight supra-orbital swelling. Œdema of interlobular tissue of lungs, and froth in trachea.   Marked enlargement of the spleen, and slight congestion of the stomach.

Case XVI. (Horse No. 55).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	75 cc.
14th	0·075 cc.	75 cc.
27th	0·2 cc.	50 cc.
40th	0·2 cc.	—
54th	0·5 cc.	—
66th	1 cc.	—
79th	2 cc.	—
92nd	5 cc.	—

There was a reaction to the blood inoculations on the sixty-sixth and seventy-ninth days, and the last inoculation provoked a fatal attack of horse-sickness, death taking place on the one-hundred-and-second day.   The maximum temperature (106·6°) was on the ninety-ninth day.

Post-mortem.—No marked change in lungs, but large amount of fluid in pericardium, and stomach much congested.   Abscess in kidney.

Case XVII. (Horse No. 56).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	50 cc.
14th	0·05 cc.	50 cc.
27th	0·2 cc.	50 cc.
40th	0·1 cc.	—
54th	0·5 cc.	—
67th	1 cc.	—
80th	2 cc.	—
93rd	5 cc. (stronger virus)	—

The last inoculation was followed by a fatal attack of horse-sickness, the temperature rising to 106° on the one-hundred-and-fifth day, and death taking place two days later.

Post-mortem.—Large amount of effusion around trachea and into pleural and pericardial sacs. Lungs very œdematous, and stomach much inflamed.

Case XVIII. (Horse No. 58).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·01 cc.	—
6th	—	100 cc.
17th	0·05 cc.	—
22nd	—	50 cc.
33rd	0·2 cc.	—
38th	—	50 cc.
49th	0·5 cc.	—
62nd	1 cc.	—
75th	2 cc. (stronger virus)	—
88th	5 cc.	—

This horse reacted to the last two blood inoculations, but survived, and its immunity to natural infection will be tested next horse-sickness season.

Case XIX. (Horse No. 61).

Day.	Dose of Horse-Sickness Blood.	Dose of Serum.
1st	0·05 cc.	—
12th	2 cc.	—
18th	2 cc. (stronger virus)	—

The last inoculation was followed by a fatal attack of horse-sickness, death taking place on the twenty-sixth day. The highest temperature (106·2°) was on the day before death.

Post-mortem.—Œdema of lungs and froth in trachea. Marked congestion of stomach.

Cases XX and XXI. (Horses Nos. 62 and 66).

In these two cases the horses were withdrawn from experiment before the dose of 5 cc. of blood was reached, being considered too valuable to undergo the risk.

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CLINICAL ARTICLES.

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A PECULIAR CASE OF CARCINOMA IN A HORSE.

By J. M'FADYEAN, Royal Veterinary College, London.

THE occurrence of carcinoma in the equine species is not so rare as to make it worth while to record the particulars of every case. The following instance, however, is so far out of the common that it appears desirable to publish a brief account of it.

The subject of it was a chestnut van horse, about ten years old, and it only came under my observation when it was handed over to
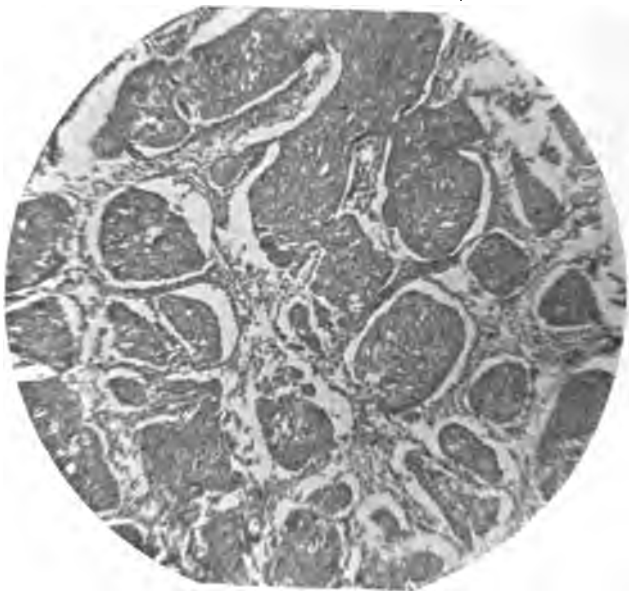

FIG. 1.
Micro-photograph of section from primary carcinoma of the membrana nictitans.

me by my colleague Professor Macqueen, in order that it might be destroyed and submitted to *post-mortem* examination. It had been sent to Professor Macqueen by Mr E. Ringer M.R.C.V.S., Leamington, as an interesting but hopeless case. The history which accompanied the horse was briefly as follows :—

Mr Ringer was first asked to see the horse on the 11th June last, on account of an enlargement in the intermaxillary space. This was at first thought to be a suppurating gland, and it was blistered, but it did not soften, and no matter could be obtained from it by puncture.

The swelling gradually increased in size, and extended upwards towards the region of the parotid gland, and downwards along the neck, until it reached the front of the chest. There was a discharge from the eyes, which was said to have been in existence for years, and Mr Ringer remarked that he thought it was caused by growths on the membrana nictitans.

As no improvement took place, the horse was turned out to grass, and remained there until he was sent to the Royal Veterinary College to be destroyed. He was admitted on the 19th November last, and an unsuccessful attempt was made to obtain some matter from the submaxillary swelling, on the supposition that the horse might be suffering from a streptothrix infection.
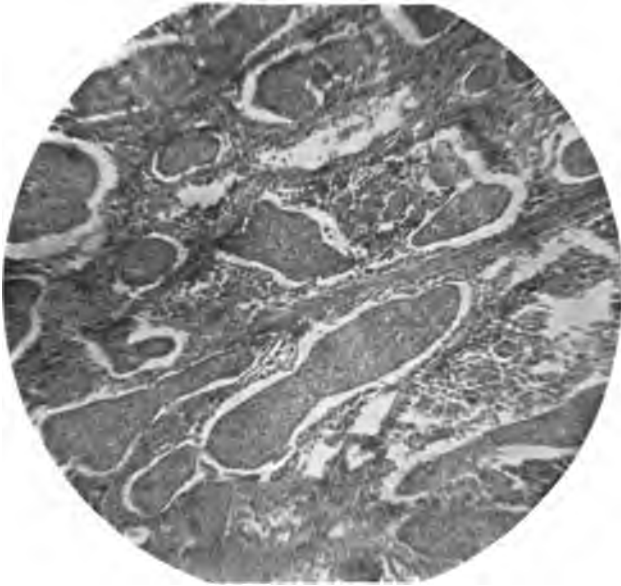

FIG. 2.

Micro-photograph of section from secondary carcinomatous tumour (propectoral growth).

The animal was destroyed on the 23rd November, and the following are the notes of the *post-mortem* examination :—

General condition good. There is a small amount of muco-purulent discharge from the left eye. The upper aspect of the membrana nictitans of that eye is covered in its anterior free part by a layer of new growth with a granular surface. This is at no part more than a quarter of an inch in depth, and its superficial extent does not amount to more than the surface of a halfpenny.

In the intermaxillary space, and mostly on the left side, there is a dense swelling, extending upwards towards the ear, and merging posteriorly into a similar dense swelling involving the under aspect of the neck and reaching as far as the front of the chest.

On reflecting the skin, that is found to be intimately adherent over

the whole of the swelling, and a quantity of yellowish clear liquid exudes from the thickened subcutaneous tissue.

On dissection the anterior lobules of the left submaxillary lymphatic gland are recognisable and apparently normal, but the packets of the posterior part are greatly enlarged and blend with a mass of new growth extending upwards under the parotid gland. On section this new growth shows a whitish ground work with numerous opaque yellowish-white specks and streaks.

The swelling along the lower part of the neck appears to be due to a diffuse fibrous thickening of the subcutaneous and intermuscular tissue, without any sign of distinct tumour formation.

On the left side of the pharynx, and apparently representing the lymphatic glands in that position, there is an irregular tumour as large as one's fist: On section this presents the same appearance as the intermaxillary growth. The right pharyngeal lymphatic glands are not visibly diseased, and the mucous membrane of the mouth, soft palate, pharynx, and larynx, is intact and apparently normal.

At the entrance to the chest, projecting three or four inches in front of the first pair of ribs, and extending backwards between these ribs to near the heart, there is a large irregular tumour, which on section resembles the intermaxillary and pharyngeal growths. This tumour closely surrounds the large vessels in the anterior mediastinum, and has contact with the trachea and œsophagus. It appears to have originated in, and to take the place of, the prepectoral group of lymphatic glands.

There is no other lesion in the thorax, and the abdominal organs appear normal.

On further dissection it was found that the growth on the left membrana nictitans had not extended beyond that structure, the eye and all the rest of the orbital contents being normal in appearance. The right eye and its appendages were also found to be normal, and, although Mr Ringer had mentioned a discharge from "the eyes," it ought to be particularly noted that when the horse was admitted to the Royal Veterinary College the discharge was from the left eye only.

For the purpose of immediate diagnosis a frozen section was made from the fresh intermaxillary growth, and this showed that the tumour was a typical squamous-celled carcinoma, having a structure identical with that shown in the two accompanying photographs.

After hardening in alcohol and embedding in paraffin, a small piece was cut from the growth on the left membrana nictitans, and this was also found to have a typically carcinomatous structure, as shown in Fig. 1. Sections were similarly prepared from the large prepectoral tumour, and a micro-photograph from one of these is reproduced in Fig. 2.

Needless to say, the chief interest in this case lies in the fact that, although the growth on the left membrana nictitans was presumably the primary one, it remained of inconsiderable size, and was at the time of death immensely exceeded in volume by the secondary or metastatic tumours.

POLYDACTYLISM IN THE OX.

By JOHN DUNSTAN, M.R.C.V.S., Royal (Dick) Veterinary College, Edinburgh.

THE specimens which I am about to describe are exceptionally well-marked instances of polydactylism. They were exhibited by me some two years ago at a meeting of the Royal Physical Society of Edinburgh, along with several other examples which I hope to describe at some future date.

FIG. 1.
The manus and pes seen from the front.

They are now in the Edinburgh Royal Museum of Science and Art, to which they were presented by Mr Taylor, who brought them over to this country from Texas, with the history that not only these two but all four limbs of the same animal presented the same abnormality, and that the dam also possessed three digits on each fore limb.

On the question which naturally arises as to whether this is a case of atavism (inheritance of characteristics from remote, as distinguished from the more immediate, ancestors), or merely dichotomy (splitting of the fœtal elements), I will not give a dogmatic opinion, but incline to the belief that it is the latter.

mesial vertical furrow, terminating above and below in a foramen. The lower one of these perforates the thickness of the bone to reappear on the posterior surface. The upper one communicates with the upper articulatory surface, as also does a similar one from a corresponding position behind.

The inner small metatarsal (? II.) extends as far down as the lower third of the large metatarsal, its tissue gradually becoming blended with it, until its existence as a separate body is no longer appreciable. It reappears, however, at the lower extremity of the bone, where it terminates by forming a small articulation for the extra digit.

The lower extremity of the large metatarsal is divided into two articulatory surfaces by a deep groove; the outer one of these is normal in shape, and the inner one has its trochlear ridge nearer to the outer side, whilst to its inner side it is closely applied the convex terminal articulatory extremity of the supernumary element, so closely in fact as to enable a single synovial membrane to suffice for the both joints.

The two weight bearing digits are normal in all respects. The extra digit resembles that described on the fore limb in all particulars except that there is a well-formed synovial joint between the second and third phalanges.

The sesamoids behind the weight bearing digits are normal. They are absent in the case of the extra digit.

The claws on the two normal digits are normal in shape and function. That on the inner digit shows no signs of having borne any weight. It again is distinctly curved towards the outer part of the foot. There are in this case also two well formed ergots.

The axis of symmetry is not deflected from its normal position, as it would be if all three digits were equally developed; on the contrary, it is placed in exactly its normal position, the extra digit giving one the impression of a bit stuck on.

INFECTIVE VENEREAL TUMOURS IN DOGS.

By the Same.

AN article under this heading appeared in the *Journal of Comparative Pathology* in March 1898, and, as the conditions described are evidently the same as those I now purpose recording, I take the liberty of borrowing the title.

The writers of the article in question, Messrs Bellingham Smith and Washbourne, mention the frequency of venereal tumours in certain dogs which they had had under observation since 1896, but are not at liberty to mention the breed. They, however, state that they have met with the condition in more than one breed.

Up to the present I have only encountered it in bull-dogs. In this particular breed I have reason to believe that it is becoming increasingly prevalent throughout the whole country, as bull bitches frequently travel from one end of Great Britain to the other for stud purposes. The reason why it is so common in bull-dogs and so rare in other breeds is that it has but little chance to spread, as the owners of bull-dogs are usually so careful of their animals of both sexes that indiscriminate service is of comparatively rare occurrence.

its middle third its position is only just indicated by the grooves before and behind. In the lower third again it stands well out, and terminates in an articulatory surface for the supernumerary digit. There are thus at the lower extremity of the bone three distinct articulatory surfaces, separated from each other by deep grooves for ligamentous attachment. As regards the two outer of these, there is little to note except that the trochlear ridges on them are not quite so distinctly elevated as in the normal ox, but on the extra one we have only a simple articulatory surface, convex in all directions, with perhaps just the slightest suspicion of the presence of a trochlea.

There are two sesamoids behind each of the normal articulations, but in the case of the extra one there is only one and that quite rudimentary.

The two outer digits show no marked deviation from the normal, but in the case of the extra one the first phalanx is only about a third the size of the normal, the second about half the size and intimately

FIG. 3.

The upper articulatory surface of the metatarsal bone with the small cuneiform bone in position.

fused to the third, which is long and slender. The outline of both these latter is perfect, but there is not the slightest sign of an articulation. Both normal digits are one inch longer than the supernumerary.

The claws on the two outer digits are normal in appearance and function. The inner claw has grown to an excessive length, curving towards the outside, but shows no sign of having ever been used as a weight bearer.

There are two rudimentary digits, each in its normal position at the back of the joint.

Right Pes of Ox.

Large Metatarsal Bone (III., IV. and ? II.)—The first thing which strikes one on examining the upper articulatory surface is the very large size of the small cuneiform, which, instead of being a small pea-sized bone, is as large as a garden bean (actual measurements, 27 mm. from front to back, 13 mm. from side to side, and 13 mm. in depth). It articulates with the large metatarsal, large cuneiform, and scapho-cuboid (see Fig. 3).

The anterior surface of the shaft of the metatarsal bone shows a

The history of the case at first did not help me very much. The dog " had not been himself" for four or five days, had been very much troubled with worms, and " had passed masses of them every day." I have seen hundreds of dogs with epileptiform fits, or with the fits which are nearly always ascribed to worms in the stomach or bowel, but this condition was quite new to me, and I hesitated to make a diagnosis. Meanwhile I gave the dog bromide of potassium in a tabloid, as he could not voluntarily swallow, and I had no means at hand for giving the drug per rectum, where I doubt if it would have been retained.

I feared the dog had been poisoned, but on that head could gain no information from the owner for a long time. At length, by judicious cross-questioning, I found out that the dog had been sick before the convulsions came on, so at once inspected the vomit. Here, amongst other matter, I was surprised to find a lump, amounting to about half ounce, of plug tobacco. On my pointing this out, I readily obtained all the information I wanted. The owner, strongly advised by some very "doggy" friends, had given the tobacco about 8 P.M. to get rid of the worms and "get a passage through him." About 20 minutes after the dose the dog had tried to defecate, and had strained violently but without success, much to his owner's disappointment. During the period of straining the dog had fallen over in convulsions. His owner thought (to use his own words) " he had strained his heart or his guts," and so had sent for me, as they were sure he was dying, and did not feel equal to tackling such a case.

The dog was now quieter but showing signs of collapse—the body cold, respiration shallow, the heart very slow and feeble. He had quite lost the use of his legs, which hung limp and paralysed when he was lifted up. The limbs, however, were occasionally slightly convulsed, while the muscles of the right shoulder showed a persistent tremor.

I had no apomorphine with me, which I much regretted, as I judged this to be a most suitable case for its use. I gave strychnine, and obtained a good reaction, the heart responding well. The convulsions ceased, after lasting for just over five hours. As soon as he could swallow he had some diluted alcohol, when he was sick again, the vomit being stained brown, smelling of tobacco, and mixed with a small amount of blood. I left him wrapped up warmly and fairly comfortable.

The next day he seemed practically well again, and ate some raw meat from my hand, and I have not seen him since.

Since visiting the dog I have looked up some of the literature on nicotine poisoning, and find it compared to that by belladonna. A case of belladonna poisoning recently came under my notice in which a fox-terrier bitch had licked herself after being freely rubbed with the B. P. liniment. In this case the symptoms came on gradually; the bitch first appeared to be going blind, stumbling over small objects in her path. She walked slowly, with difficulty. and tended to move in a circle, finally staggering and falling with complete loss of muscular power in her hind legs only. There were no convulsions, but persistent grinding of the teeth ; pulse and respirations greatly accelerated. The symptoms gradually subsided, and passed off in about four hours after the administration of small quantities of brandy in

warm milk. The pupil was not dilated as one would have been led to expect. In fact, it was if anything contracted.

In the above case of nicotine poisoning the condition of the eyes was peculiar. The globe was so retracted that the membrana nictitans obscured the cornea, and only an imperfect view of the iris could be obtained. So far as I could judge, the left pupil was slightly, and the right pupil distinctly, contracted. In writing of nicotine poisoning in human beings, Hale White says the pupil is contracted by sympathetic paralysis and irritation of the 3rd nerve. Dr Thomas Stevenson, the poison expert, states that the pupil is widely dilated.

If one were in any doubt, however, as to which of the two poisons is in the system, conclusive evidence should be afforded by the mouth. which in the case of belladonna would be dry or gummy, whereas in the case of nicotine poisoning the mouth would be moist, or even contain a quantity of saliva and mucus, and probably smell of the tobacco.

A DIFFICULT DIAGNOSIS.

By ROBT. G. ANDERSON, M.R.C.V.S., Wellington, Somerset.

ON 12th March last a six-years-old Devon cow in full milk was seen by its owner to be unwell, and it was attended by me on the same day. Temperature 106°, pulse frequent but regular and of good volume. A cause for this fevered state seemed to exist in an inflammatory condition, evidenced by a marked dulness, but without pleuritic sounds, at the base of the right lung. A marked grunt suggested an implication of the liver and second or third stomach. Without previous history of indigestion, a foreign body was not suspected.

Treatment here, as throughout the duration of the case, was directed solely to the alleviation of marked symptoms.

A previous experience of redwater in a similar animal caused the owner to be on the look-out for its symptom, and on the second day he had noticed a flow of distinctly coloured urine. This symptom disappeared in a few days under specific treatment.

At this time the condition of the lung had improved, the grunt was also gone, but the temperature remained high. Auscultation to discover if any fresh portion of lung had become invaded, seemed to find it high up on the left side, but more particular attention showed the vesicular murmur to be of its normal softness, with slightly increased frequency, and the quite marked roughness to proceed from the tissues external to the ribs. Indeed, a movement of the ear over the part, and further manipulation of it, made out an emphysematous condition of the subcutaneous tissues. This was also to be distinctly felt over the loins. It was thought possible that this condition was due to a puncture of lung or windpipe, but its probability was doubted, and indeed eventually dismissed. In a district where quarter-evil is well known this symptom is taken as decisive of its existence, but the age of the animal and the length of time that it had lived after showing this symptom were difficult to get over.

To end what seemed to be a very uncertain case, and solve the mystery of its causation, slaughter was advised. A second opinion was first had, and this being to the effect that the condition was

probably one of punctured lung, which need not of course be regarded hopelessly, treatment as suggested was carried out. A slight improvement also took place at this time, and the temperature dropped to 103°. But it was a deceptive rally, as the grunt reappeared and the dulness over the base of the right lung persisted in spite of repeated counter-irritants. Little change was now noticeable from day to day, except a slightly increasing weakness and progressive sinking of the eye. The subcutaneous emphysema, accompanied only in the left lumbar region by swelling, appeared along the whole length of the upper part of the neck on the left side, and also over the ribs on the lower right side, but much anterior to the part which had been mustarded. Iodine solutions were injected into these parts, and mixed blisters of cantharides and hydrarg. biniod. were rubbed in where they persisted. This caused their disappearance from some parts. It is to be noted here that this emphysema did not seem to proceed and extend from one point, nor were the parts showing it continuous, or situated where a lung lesion could be diagnosed, or where counter-irritants had been applied. Lung symptoms manifestly got worse, the grunting increased, and appetite had quite gone. The length of time this cow kept feeding and was able to get up and walk about was remarkable.

The case could only be diagnosed as inflammation of the right lung, with functional or organic derangement of the stomach.

The animal died after an illness of three weeks' duration.

Post-mortem.—On removing the skin the subcutaneous tissue discharged fluid blood. Only over the left lumbar region, where the emphysema had persisted, was there discoloration, and over the ribs, high up on the right side, where nothing had been felt nor had blisters been applied. This was of an identical appearance with the lesions of quarter-evil. The muscular tissue at this part, except near the surface, was healthy, and at no point was there evidence of communication with air passages or cells. In the abdomen, only the second stomach showed a peeling of its mucous membrane.

In cutting out the lungs a large bronchial lymphatic abscess was evacuated. The lungs did not collapse, and the right showed its surface raised by nodules in its substance. These were bronchi filled with inspissated matter and cavities filled with the same ; lung tissue blackish between these nodules. The left lung showed these lesions lesser in number, and its colour was nearly normal. The heart was healthy except that its muscular tissue was soft and flabby.

There was no trace of a foreign body.

I was inclined to the opinion that a foreign body, not discovered, was at the bottom of the mischief, and that the lung nodules were perhaps due to the irritation of gruels, etc., which had passed into the lungs. But a microscopical examination of these nodules was first desirable, and this was made by Professor M'Fadyean. He reported that the lung nodules appeared to have been invaded by the necrosis bacillus, and thought the case a rare one if no similar lesions were present in connection with the stomachs or liver. He also thought that the emphysema of the subcutaneous tissue must have originated in some laceration of the lung tissue or defect in the trachea.

There were no lesions in the liver or rumen, and I am therefore inclined to think that the emphysema had a bacterial origin.

Abstracts.

CHRONIC PERIARTHRITIS OF THE HOCK IN HORSES.

THIS disease is by no means so uncommon as is generally believed. During the last few years about one-half per cent. of all horses suffering from external diseases, brought to the Clinique for the larger domestic animals in Berlin, were found to be suffering from it. From the etiological point of view two or even three varieties of periarthritis may be distinguished, viz., primary periarthritis, secondary periarthritis, and a third form, mentioned by Gossman as due to inflammation of the hock, and consisting in secondary periarthritis. From the pathological and anatomical point of view it is important to note that the periosteum and bone are principally affected.

Primary periarthritis is externally indicated by thickening of the joint, the thickening being of bony hardness and closely adherent to the surface of the bone. On removing the superficial soft tissues that portion of the bone immediately beneath the periosteum is seen to be covered with osteophytes. The rarefying ostitis is replaced in the bony tissue by osteosclerosis. The periosteum covered with osteophytes is at first soft and easy to cut, but as ossification proceeds it becomes very hard and resembles ordinary bone tissue. The surface of the bony new growths is smooth ; the growths them-selves vary greatly in size. They most commonly affect the tarsal bones, but may also extend to the distal end of the tibia and the proximal end of the metatarsus. The bony tissue also contributes to the formation of the osteo-phytes ; the capsular ligament, synovial membrane, and lateral ligaments seldom show more than a certain amount of thickening, and are never con-verted into bone except at their points of insertion. The tendons and sheaths in the neighbourhood of the tarsus are little altered. Gossman never saw ossification of tendons or tendon sheaths; the skin and subcutaneous tissues were often thickened, and occasionally showed softened areas, but the articular surfaces were never diseased. Extension of the primary peri-arthritis to the articular surfaces and spavin formation were never observed.

Secondary periarthritis follows inflammatory processes in and about the tarsus. Both the periosteum and sub-periosteal bony tissue are implicated in the formation of the exostoses. Microscopical examination shows that the process may begin either in the sub-periosteal bony tissue or in the peri-osteum itself; usually the primary disease consists in inflammation of the bony layer immediately beneath the periosteum. In consequence of the inflammatory changes the periosteum and sub-periosteal bony tissue become firmly connected. When the disease starts in the periosteum itself it is accompanied by marked dilatation of the blood vessels and extensive cellular infiltration. The process soon extends from the periosteum to the bony tissue.

The most striking feature of the disease is the increased size of the joint. The exostoses sometimes affect only the surface of the tarsal bones or of the talo-crural or tarso-metatarsal joint. In most cases the outlines are sharply defined. The surface of the deformed joint is usually smooth, and the skin covering it is tensely stretched and firmly adherent.

Lameness of varying type occurs during the course of the acute inflam-matory processes. At first the diseased limb is rested on the toe, the joints are flexed, and the leg is allowed to hang loosely from the thigh, the body weight being transferred to the other limb.

When both hocks are diseased the front legs are brought under the body in order to relieve the hind of weight. When the process has become arrested or the thickening is inconsiderable, or when periarticular anchylosis of the hock has occurred, lameness may be absent. Extensive thickening may mechanically interfere with the flexion and use of the joint. When there is mechanical interference with movement of the joint the degree of lameness is principally determined by the degree of existing periostitis. The above-described symptoms may be modified by the existence of complications like spavin, the inflammatory form of curb, ringbone, arthritis, etc. The condition must be distinguished from bruises and sprains of the hock joint, acute peri-arthritis and periarticular inflammation, ringbone, curb, fibrous thickening of the skin around the hock, dilatations of the synovial capsules and tendon sheaths in the neighbourhood of the hock.

The prognosis should be reserved, as the termination is doubtful.

The chief indication in treatment is rest.

Bearing in mind the nature of the disease, it is clear that recovery can seldom be complete, though the inflammatory changes can often be cheeked and the animal again rendered useful. The animals should be rested. Treatment consists of cold applications, followed at a later stage by warm fomentations and massage, with soothing lotions or slightly stimulating oint-ments. The use of blisters or firing is contra-indicated, the chief require-ment being complete rest of the part; blistering does not achieve this object, and is apt to increase the inflammation and its attendant consequences. As a last resort the tibial and peroneal nerves may be divided. (Gossman, *Monats. fr. Tierhlk.*, Vol. XV., Parts 9 and 10, and *Berl. Tierärztl. Wochens.*, No. 36, 1904.)

A REPORT ON POLYVALENT SERA WITH SPECIAL REFERENCE TO IMMUNITY AGAINST THE ORGANISMS OF SWINE PLAGUE.

As a result of earlier experiments, Wassermann and Ostertag had concluded that the individual varieties of the organism of swine plague show biological differences of such importance as to require being carefully borne in mind when preparing serum for general use against that disease. The so-called polyvalent swine plague serum is prepared in accordance with this theory.

The polyvalence aimed at in connection with swine plague and "white scour" of calves differs from that obtained in connection with streptococci. In preparing antistreptococcic serum, streptococci obtained from widely differing cases of streptococcus disease were employed, and the serum so prepared proved active against all the different clinical forms of streptococcus disease In contra-distinction to this, the authors came to the conclusion that in-dividual varieties of one family of micro-organisms, all of which, clinically and epidemiologically, produce one and the same disease, nevertheless show variations when used for purposes of immunisation, and that these variations must be taken into account when preparing the serum.

The bacterial protoplasm of many species of micro-organisms appears to consist of a large number of different principles. This fact has been demonstrated by Wassermann and Ostertag in respect of swine plague, by Jensen as regards "white scour," and by other authors in relation to a large number of other organisms.

The structure of the individual varieties of the swine plague organism does not vary greatly; in all, the properties of the greater part of the protoplasm are identical (dominant receptor). The dominant receptor determines the peculiarities of the species. This is confirmed by the observation that a

monovalent swine plague serum, *i.e.*, one active against a particular variety of the swine plague organism, although in practice possessing insufficient activity against other varieties, nevertheless exercises a certain influence.

This dominant receptor is accompanied by a series of subsidiary receptors of varying composition. The existence of subsidiary receptors explains why a monovalent serum proves insufficiently active against varieties other than that with which it was prepared.

In regard to swine plague, one must remember that the virulence of the different varieties for experimental animals bears no direct relation either to their virulence for swine or to the protective power of the serum prepared through their agency. For instance, a monovalent serum obtained by the use of a highly virulent variety may only be able slightly to delay death, and may prove of no value against another variety which in itself is much less virulent. The view that by using a single extremely virulent variety of swine plague organism a serum might be prepared which would have an equally valuable protective influence against all other varieties, is regarded by Wassermann and Ostertag as erroneous. Moreover, attempts to cultivate a single variety so that its receptor apparatus should correspond with those of all other varieties are misplaced, single species of micro-organisms retaining their peculiarities very stubbornly. Nor in practice is it sufficient to immunise different animals with one and the same variety, and to mix the sera so obtained in order to average the biological peculiarities of the species. In dealing with a disease in which the organism shows such great and numerous variations, it is much more important to utilise as many different varieties of organism as possible in preparing the serum.

When a monovalent serum is utilised against a variety of organism whose subsidiary peculiarities it completely suits, a smaller quantity is required than of a polyvalent or multi-partial serum, but when, on the other hand, it meets with a variety to whose dominant receptor alone it corresponds, without sufficiently affecting the individual and varying subsidiary peculiarities, the monovalent serum confers a certain degree of protection, which, however, in practice proves insufficient.

On the other hand, the polyvalent (multi-partial) serum must be employed in somewhat higher concentration against individual varieties, but if sufficiently polyvalent there is hardly a variety against which it cannot confer practical protection, in consequence of its greater richness in the varying secondary products. Neisser and Wechsberg's further experiments show that polyvalent sera contain a much greater variety of secondary products as compared with monovalent sera.

Under all practical conditions polyvalent swine plague serum, being so much richer in the secondary products, is much to be preferred, and exercises a more uniform protective influence against the varying forms of disease to be met with throughout Germany. (Wassermann and Ostertag, *Zeitschr. f. Hyg. und Infektionsk.*, Vol. XLVII., Part 3, and *Oesterr. Monats. fr. Tierhlk*, Nov. 1904.)

AN EXPERIMENTAL CONTRIBUTION TO THE QUESTION OF IMMUNITY AGAINST SWINE PLAGUE.

WHEN it became clear that active immunity against swine plague could not be attained, attempts were made to procure passive immunity. It was found possible to produce protective sera by treating animals with the bacteria of swine plague, and commercial forms of such sera were prepared by Bruck under the name of "Septicidin," and by the Höchst Farbwerken. No great practical success, however, was attained.

The cause of these failures was explained by Wassermann and Ostertag. They showed that a serum obtained by the use of one variety did not exhibit sufficient protective power against all other varieties of the same species, but only against the variety which had been used to immunise the animal yielding the serum. The virulence of the variety employed is not of primary importance, for serum prepared from a very virulent variety showed no protective action against varieties of very much less virulence. According to Wassermann and Ostertag, the explanation of this peculiarity was to be sought in ultimate biological differences between the different organisms of swine plague, and particularly in the fact that the bacterial protoplasm did not consist of a single substance but was built up of different components. In the different varieties these components might vary considerably, and thus cause important differences, inasmuch as each component produced special peculiarities in the protective serum obtained by its injection.

Bruck studied the principle of polyvalence more thoroughly by experimental methods.

As a result of parallel tests of the before-mentioned "septicidin" and Höchst swine plague sera, Bruck proved that these sera did not confer protection against all varieties, however virulent the variety employed in preparing them, but only gave protection against some, whilst the polyvalent swine plague serum prepared by Wilhelm Gans (Frankfurt) protected against all varieties alike, even when the dose of bacilli administered was greatly in excess of the (usually) fatal quantity.

Bruck came to the following conclusions :—(1) The virulence of the swine plague organism undergoes great changes by artificial cultivation. (2) The swine plague organism forms no toxin soluble in water. (3) The bacillus suisepticus does not form any considerable amount of haemolysin or leucocidin (substances causing disintegration of red and white blood corpuscles). (4) In practice polyvalent sera are greatly to be preferred for conferring protection against swine plague. (5) Polyvalent is distinguished from monovalent serum by the fact that in consequence of its greater richness in varying secondary products a somewhat higher concentration is required, but it affords protection over more widely separated districts.

The value of the principle of polyvalence as applied to the preparation of swine plague serum must, in view of the success obtained, be regarded as fully demonstrated, for when a monovalent serum is employed against a variety of bacillus to whose secondary peculiarities it is unfitted it must fail, whilst the chances of success in using a polyvalent serum are, as above demonstrated, much greater. (Bruck, *Zeitschr. f. Hyg. und Infektionsk*, Vol. XLVII., Part 3, and *Osterr. Monats. fr. Tierhlk.*)

EXPERIMENTS WITH SEPTICIDIN IN THE TREATMENT OF SWINE PLAGUE.

BREIDERT tested the activity of the septicidin prepared at Landsberg. As sent out, it is stated to give protection against swine fever, swine plague, fowl cholera, and pneumonia of calves (chronic form of "white scour"). He used ten varieties of swine plague bacteria, a fatal dose of which was rendered harmless by injecting ·01 gramme of Wassermann and Ostertag's polyvalent serum. Septicidin only afforded protection against one of these varieties. The general conclusion was that septicidin gave no protection against eight selected varieties of swine plague organism, whilst Wassermann and Ostertag's polyvalent serum protected against all of them.

Breidert also tested septicidin against swine fever and fowl cholera, and found, contrary to the claims made for it, that it conferred no immunity. (Breidert, *Zeitschr. f. Hyg. und Infektionsk*, Vol. XLVII., Part 3, and *Osterr. Monats. fr. Tierhlk.*)

DISEASES PRODUCED BY TRYPANOSOMATA.

Professor Koch recently delivered an address, of which the following is the substance, to the Berlin Medical Society regarding his experiences and observations on diseases produced by trypanosomata in Africa :—

A wide field of study has recently been opened by the discovery of various pathological protozoa. Three discoveries especially have directed attention to these special disease organisms—

1. Laveran's discoveries regarding malaria. Ross has shown that the malaria parasites are carried by mosquitoes (anopholes).

2. The discovery of the protozoa of Texas fever by Smith. In this case ticks (ixodes bovis) convey the disease.

3. The discovery of the trypanosoma of the tsetse disease, which is conveyed by a stinging fly (glossina morsitans).

These discoveries were followed by numerous others indicating protozoa as causes of disease.

The trypanosomata are morphologically distinguished by the existence of a flagellum. When fresh blood is examined it is scarcely possible to overlook the protozoa in the preparation, for attention is at once attracted by the energetic way in which the red blood corpuscles are continually being displaced. The peculiar form of the protozoa, however, can only be detected in stained preparations. Romanowsky's staining method is probably the best. Trypanosomata stained by this method show a fish-shaped body, the front end of which carries a flagellum. The body of the trypanosoma is coloured blue. At the anterior end may be seen a red-stained nucleus ; at the opposite end a much smaller red spot, which has been termed the nucleolus, but is more properly described as the centrosome. From the centrosome a red thread extends along the outer margin of the body as far as the front extremity, where it becomes continuous with the flagellum.

The trypanosomata increase by longitudinal fission. The centrosome and then the nucleus divide, and finally a second flagellum is formed. Sometimes the young trypanosomata remain connected, producing the so-called "rosettes."

The disturbance produced by trypanosomata seldom becomes acute. but often continues for years. The only sign of disease consists in ill-defined fever with long intermissions. The destruction of the red blood corpuscles causes anæmia, the animals or men become weak and waste away, œdema and sometimes erythema occur at varying points in the body, and occasionally the lymphatic glands become swollen.

Trypanosomiasis of rats is one of the most frequent forms of the disease. About 30 per cent. of all rats harbour trypanosomata, and in some cases the percentage is as high as 90.

The trypanosomata of rats are morphologically distinguished by possessing a long point at the posterior end—*i.e.*, the end opposite the flagellum. The centrosome lies rather far back.

It is peculiar that the trypanosomata of rats seem to produce no morbid symptoms, nor do they injure their hosts in the slightest degree. They can only live in the bodies of rats, and not in those of mice. Conveyance from

2 A

infected to healthy rats has been shown by Rabinowitch and Kempner to occur through the medium of fleas.

The classical land of the tsetse disease is in the neighbourhood of the Zambesi. There it was seen and very well described by Livingstone; but, unfortunately, further investigations have shown that tsetse disease extends over the whole of Africa.

In contrast with the trypanosoma of rats, that of tsetse disease exhibits a distinct rounding off of the hinder end. The centrosome also lies very far back. Whilst the trypanosomata of rats can only be conveyed to the one species those of tsetse disease thrive in all mammals, particularly in the horse, mule, ox, dog, rat, and mouse.

The tsetse organism has been shown to kill both horses and mules, but to be less dangerous for oxen. A certain relative immunity exists in some races. As regards the ass, observers are not agreed; Koch failed to infect it. Sheep and goats are also but slightly susceptible, and, luckily, man is quite insusceptible. The conveyance of trypanosomata from the blood to uninfected animals occurs through the medium of a stinging fly (the glossina morsitans).

Surra is endemic in the Phillipines, Java, and the island of Mauritius. Koch regards the trypanosomata of surra as strictly analogous with the parasites of tsetse disease. Horses (and, in India, elephants) especially suffer from surra. Although the glossina morsitans does not occur in India, other stinging flies replace it and convey the disease.

Another variety of trypanosomiasis is "mal de caderas," seen in South America, particularly in Argentina and Brazil. It affects horses. According to Koch, the parasites of "mal de caderas" exactly resemble the tsetse and surra parasites. Other observers, however, declare that the "mal de caderas" parasites are distinguished from those before mentioned by their particularly small centrosome. "Mal de caderas" affects not only horses, but all the other animals which suffer from tsetse.

Another variety of trypanosoma, the trypanosoma Theileri, is especially striking on account of its size. It is only found in oxen, and exhibits a very slight degree of virulence.

In 1901 trypanosomata were first recognised as human parasites by Castellani, who found them in the cerebro-spinal fluid. Ross regarded this trypanosoma as the cause of sleeping sickness. The parasites of human trypanosomiasis and of sleeping sickness are identical.

Koch declares that sleeping sickness, which always ends fatally, is only to be regarded as the terminal stage of human trypanosomiasis. How long the trypanosomata may continue to exist in man without producing evident symptoms of disease is shown by the fact that negro slaves have died of sleeping sickness five years after leaving Africa. The parasites of sleeping sickness are morphologically indistinguishable from those of surra and tsetse disease. Koch divides trypanosomata into two great groups.

The grouping is based on three important peculiarities: firstly, the morphology of the parasite; secondly, its virulence; and, thirdly, its relation to the host.

The first group only exist in one species of animal. They have become so completely accustomed to this method of life that they cannot exist under other circumstances. Their virulence is slight but constant. This group comprises the trypanosoma of rats and the trypanosoma Theileri.

The second group (to which all other trypanosomata belong) shows great variation in virulence and in form. These trypanosomata are not peculiar to any one species, but may affect dogs, rats, horses, etc. Their morphological peculiarities also vary according to the animals in which they are found. Thus, the tsetse parasites when cultivated in the bodies of dogs and rats become much smaller than usual, and the centrosome appears near the end;

when cultivated in horses the end appears pointed, and the centrosome lies near the centre; in the pig the parasites lose their peculiar short flagellum. Their virulence also varies within wide limits.

Some time ago the Berlin Zoological Gardens received a stallion and a mare from Togoland. During the journey these animals had to pass through a tsetse-infected district. They became infected, and four months later Veterinary Surgeon Dr Martini in Berlin found trypanosomata in their blood. Koch states that it is sometimes very difficult to discover the organisms in the blood, particularly when they are present only in small numbers. In such cases susceptible animals must be inoculated with several cubic centimetres of infected blood and the results awaited. All the dogs infected with blood from the Togoland stallion died, whilst nine dogs injected with blood from the mare resisted. The stallion died in the Institute for Infectious Diseases, but the mare lived for a year, at the end of which time Koch injected her with trypanosomata from the stallion; soon afterwards she succumbed. The virulence of the trypanosomata from which the stallion and mare respectively suffered therefore seems to have been very different.

It has been found possible, as in the case of bacteria, to modify the virulence of trypanosomata by successive passages through different animals. By inoculating dogs with comparatively innocuous trypanosomata and conveying the disease from dog to dog the virulence is markedly increased. On the other hand, parasites which prove very virulent for oxen become much less active for these animals after passages through rats and dogs. This apparently trifling discovery laid the foundation for protective inoculation experiments. Parasites of the second group can also exist in the bodies of almost all mammals.

Koch is of opinion that the parasites of surra in India and of tsetse disease in Africa are absolutely identical. Laveran, on the other hand, states that he has protected animals against tsetse, and that they have nevertheless suffered from surra.

That this in no way disproves the identity of the two parasites is best shown by the experiments on the Togoland horses, for both were infected with the same parasite; the stallion died and the mare lived, nevertheless the mare also died when infected with parasites from the stallion.

Koch, whilst in Dar-es-Salaam, made some interesting experiments for the purpose of discovering a method of protective inoculation. He had found that the virulence of the ox parasites could be modified. He therefore inoculated oxen first with these weakened parasites and afterwards with others of high virulence. All the control animals died while those treated as above remained alive.

Veterinary Surgeon Schmidt kept these animals under observation, and reinoculated them from time to time with highly virulent material, notwithstanding which they were still perfectly well six years after the first inoculation.

In practising this method, however, the trypanosomata used for the first inoculation must not be unduly weakened. The method would have appeared fully successful were it not for the fact that the protected and apparently quite vigorous animals still suffered from the presence of parasites in the blood. To extend its use, therefore, meant that one would not suppress but would spread the disease. The effect would be to produce herds harbouring the parasite, and which herds, though exhibiting no signs of illness, would nevertheless in a sense be propagating the active cause. Further observation has also shown that the protection so conferred is only relative. Dogs can always be infected with the blood of such animals. It has long been known in Africa that antelopes and buffaloes harbour trypanosomata in their blood without showing external signs of disease.

Another method of protection must therefore be sought, such as destroying

the various stinging flies; but this offers little hope of success. Koch admits that he sees no method of dealing with them. The other method is di·ected against the parasite, and here he seems more hopeful. The disease can be rooted out by killing all diseased animals suspected of disease. The line of procedure is indicated by the experience gained in Mauritius and Java. When surra broke out in Mauritius almost all the oxen died in two years. In Java the nature of the disease was early recognised, and all suspected animals were at once slaughtered or isolated until slaughtered; in this way the disease was soon stamped out.

Trypanosomia-is in man necessarily cannot be dealt with in this way. Nor, unfortunately, is there any known drug which in this disease acts after the manner of quinine in malaria. The only materials which until now have given good results are arsenic and a dye-stuff called trypan red. Whether they will prove fully successful cannot yet be determined. Koch calls for common investigation by all nations having colonies in which trypanosoma diseases occur. (*Berl. Thierärztl. Wochens.*, No. 45, p. 736).

THE TRANSMISSION OF TUBERCULOSIS FROM ANIMALS TO MAN THROUGH THE MEDIUM OF MILK, AND ITS PREVENTION.

LONG before the discovery of Koch's bacillus Gerlach (1860) and Klebs (1873) proved that the milk of tuberculous cows was virulent. Many investigators, including Nocard, Galtier, Hirschberger, Gebhart, Rabinowitch, Kempner, etc., afterwards demonstrated the presence of Koch's bacillus in milk from certain tuberculous cows.

The first proposition definitely laid down was that Koch's bacillus was always present in the milk of cows suffering from tuberculosis of the udder.

In France statistics show that tuberculosis of the udder is somewhat frequent. Of 675 tuberculous cows distributed over seven of the central and south-eastern departments, 44 or 6·5 per cent. showed mammary lesions. Of 1481 cows slaughtered in the abattoirs of the same regions 79 or 5·3 per cent. suffered in a similar way. In Isère, where animals are regularly inspected, 14 cases of tuberculosis of the udder were found among 121 cows—a pro-portion of 11·5 per cent. In 1902 Huon at Marseilles found 21 cases of tubercu-losis of the udder among 698 tuberculous cows; Labally 10 cases amongst 166 tuberculous cows slaughtered at Saint Etienne. At Toulon, of 103 tuberculous cows 15 were found to be suffering from mammitis, and at Grenoble Blane found 5 cases of mammitis amongst 17 cows.

The danger nevertheless would be relatively slight if the dangerous milk were only furnished by cows showing clearly marked external lesions of the udder.

Unfortunately this is by no means the case, and numerous observations prove that milk from apparently healthy animals may prove virulent. The experiments of Rabinowitch and more particularly of Mohler are very con-vincing in this respect. Of fifty-six cows which reacted to tuberculin thirteen gave infected milk. Nevertheless none of these animals when examined after death (which in some cases was deferred for a year after the first test) presented tuberculous lesions of the udder. All exhibited more or less tuberculous lesions, but only two showed definite clinical symptoms.

Professor Moussu of Alfort, who undertook some experiments for the

purpose of checking Rabinowitch's and Mohler's statements, also obtained very interesting results. Of sixty animals which reacted· to tuberculin, but showed only trifling clinical signs of the disease, nine were found to give infected milk. On *post-mortem* examination two showed clearly marked tuberculous lesions of the mammary lymphatic glands, but no trace of tuberculous lesions in the parenchyma of the mammary gland itself.

It must therefore be admitted that the milk of cows suffering from tuberculosis may contain bacilli of that disease even although the udder itself shows no appreciable clinical lesions.

The next question which arises is that of the degree of danger from swallowing milk containing tubercle bacilli.

Although the ingestion of meat furnished by tuberculous animals is not very dangerous, the consumption of raw milk containing large numbers of bacilli is an exceedingly serious matter. Despite all that has been said by Koch and the supporters of his theory regarding the essential difference between human and animal tuberculosis, medical literature contains very convincing descriptions of the transmission of bovine tuberculosis to man through the medium of the milk.

Moreover, the very large number of investigators who have checked Koch's assertions have proved that as a general rule the bovine tubercle bacillus is by far the most virulent for all species of mammals.

The writers therefore refuse to confirm Koch's dictum that bovine tuberculosis does not constitute a danger for man. At the last International Congress of Hygiene held at Brussels in 1903, it was decided that precautions should be taken to prevent tuberculosis being transmitted from animals to man.

It is perhaps scarcely necessary to repeat the observations of Thorne-Thorne, who showed that in England, where, in consequence of the sanitary measures which have been put in operation, the general mortality from phthisis has diminished by 45 per cent. since 1850, the mortality from abdominal tuberculosis in children less than one year old has increased since that date by 27 per cent. Thorne-Thorne declared that this increase in infantile mortality from tuberculosis was due to the absence of all control of dairies and of any law preventing the sale of milk from tuberculous cows.

The extreme frequency of tuberculosis in pigs fed with milk refuse shows the degree of danger of infection by milk. In the United States, where pigs are exclusively fed with grain, only 579 pigs out of 13,616,539 were found to be tuberculous during the years 1894-95, that is to say, a percentage of ·0004. In Denmark, on the contrary, where pigs are fed with skim milk, the proportion of tuberculous animals rises to 15 per cent. The pigs kept by the dairy owners at Danzic are diseased in as high a proportion as 60 to 70 per cent.

Even in the absence of such proofs, the experiments made by Nocard and Gratia on monkeys, whose organisation so closely resembles that of man, would justify alarm.

Nocard, having shown that the monkey is much more readily infected through its digestive tract with bovine bacilli obtained from cases of tuberculous mammitis than with human bacilli, carried out the following experiment, which hitherto has remained unpublished. On the 5th March 1903, two monkeys (Macacus Rhesus) were given a small quantity of rice containing 10 cc. of milk from a cow suffering from tuberculous mastitis. One died on the 31st May, the other on 20th September 1903. Both showed on *post-mortem* examination extensive lesions of intestinal tuberculosis.

At the Hygienic Congress held at Brussels in 1903, Gratia described an experiment on three monkeys to which he gave in three to five portions 80 grammes of milk obtained from cows showing lesions of mammary tuberculosis.

They died with grave lesions of abdominal tuberculosis after an average period of 126 days.

Infected cows' milk seems particularly dangerous because it is usually consumed in a raw state, because it is the regular, or even the exclusive diet in certain diseases, and sometimes in health, whilst many children receive nothing else. It is unquestionable that such repeated infection greatly favours the chance of contracting the disease, particularly in young children.

Behring and Röhmer have recently shown how easily tuberculous infection through the bowel occurs in young children. Behring goes so far as to declare that babies are specially inclined to contract tuberculosis through the intestine because in them the mucous membrane still lacks certain protective mechanisms which exist in the adult and which check the passage of the pathogenic germs into the tissues and lymphatic channels. The danger of the transmission of bovine tuberculosis to man through the medium of milk is beyond question. although it threatens adults much less than infants.

There is no doubt, however, that milk containing numerous bacilli can be consumed with absolute impunity provided it be previously boiled. for the presence in the milk of toxic products produced either by the diseased animal's tissues or by the tubercle bacilli themselves is not then dangerous. Galtier's experiments are very clear on this point. Unfortunately the custom of drinking raw milk is still widely prevalent, and even when boiling is practised it is often carried out imperfectly and is not invariably efficacious.

Measures ought therefore to be taken to remove the idea that boiled milk is less nutritive than raw milk and that it is less easily digested. The advantages of only drinking boiled milk, in which any dangerous germs have necessarily been destroyed, should be steadily impressed on the public.

The practice of sterilising milk is nowadays widely accepted, particularly in the feeding of children. Even though boiled milk were not quite so suitable as raw milk, and had not all the properties of the latter, it certainly possesses all the nutritive qualities of raw milk; and, if one bears in mind the great practical advantages of sterilising milk, the trifling drawbacks are scarcely worth attention. To sum up, in the opinion of the authors, the following propositions may be regarded as now completely demonstrated and suitable as a basis for a system of prophylaxis :—

Bovine tuberculosis is transmissible to man.

The milk of tuberculous cows is often virulent, particularly when the animals are severely attacked. That of cows suffering from tuberculosis of the udder is always very dangerous.

The danger of transmission of bovine tuberculosis to man by milk cannot be questioned.

Provided it be properly boiled dangerous milk becomes innocuous.

It is therefore necessary to establish a system of inspection and control of the milk traffic for the purpose of safeguarding public health.

Abroad some interesting experiments have already been made with this object. At Copenhagen the sale of milk has been controlled since 1890, and severe measures have been taken with regard to milk from tuberculous animals. In Schleswig-Holstein, Baden, Danzic, and Sigmaringen, the sale of milk is very severely regulated. In certain German towns milk is examined in the markets and milk shops by special inspectors. At Dresden and Frankfort-on-the-Oder (1900), the milk businesses have been placed under the sanitary authorities. In Prussia inspection of the dairies where nursery milk is produced was made obligatory in 1900. The sanitary veterinary surgeons have permanent access to the Berlin dairies, and must visit all these establishments at least once in every three months. Milk from animals showing clinical signs of tuberculosis may not be sold.

In Italy measures were passed in 1890 and in 1901 regulating the trade in

milk. Similar laws exist in Belgium, Switzerland, England, and in the majority of the States of North America.

Two different systems have been formulated to protect the public from the danger of consuming raw milk containing tubercle bacilli. One consists in pasteurisation or sterilisation, the other in forbidding the use of milk from tuberculous cows. The latter system necessitates the control of the milk trade, the regular testing of milch cows with tuberculin, and the sanitary inspection of dairies.

Pasteurisation of Milk.—In this case the milk should systematically be pasteurised by the dairy companies. Provided the operation is properly carried out in closed vessels at a temperature of 85° C., the germs are destroyed, whilst the flavour of the milk is little affected and its keeping qualities are greatly improved. Pasteurisation has been made obligatory throughout Denmark, and has been regularly carried out since 1898, to the entire satisfaction of the authorities and of the public. This system necessarily calls for the creation of a staff of inspectors to see to its proper execution and to prevent fraud.

Although pasteurisation when properly carried out renders dangerous milk safe, it may prove ineffective if carelessly performed. Moreover, the custom of consuming milk which is sold without any guarantee of cleanliness, etc., will still continue for a long time, and it therefore becomes necessary to undertake measures for the protection of the consumer.

Inspection of Milk when exposed for Sale.—Although the commercial inspection of milk is valuable for detecting changes in its chemical composition, and although in a large measure it prevents fraud, it cannot obviate the danger of commercial milk containing tubercle bacilli.

The discovery of tubercle bacilli in milk necessitates either microscopical examination or inoculation of guinea-pigs.

The first of these methods is laborious and uncertain. Either the bacilli escape notice, or the milk, even when carefully collected, is found to contain acid-fast bacilli which cannot immediately be distinguished from those of tuberculosis.

Inoculation of guinea-pigs is the only reliable method of testing, but it necessitates a delay varying between several days and a fortnight. Moreover, in most cases it would be impossible to trace the source of the germs even when they were discovered and to prevent recurrence, considering that commercial milk is almost invariably a mixture obtained from different farms.

Commercial control is therefore insufficient to prevent the milk of tuberculous animals being sold.

The Tuberculin Test for Milch Cows.—This test, if properly carried out, would in itself suffice to prevent any tuberculous milk being supplied to the public. From the scientific point of view, therefore, the tuberculin test, if systematically practised, constitutes the best mode of preventing tuberculosis being spread through the medium of milk.

The tuberculin test would also render immense service in preventing the spread of bovine tuberculosis. It is harmless, inexpensive, and permits of all the tuberculous animals in a dairy being rapidly detected. These should be separated from the healthy ones and prepared for slaughter. After completely disinfecting the buildings, the owner need only test all newly-bought animals with tuberculin in order to guard himself against any further loss.

Unfortunately, almost unsurmountable difficulties arise as soon as this system is made obligatory. The operation lends itself to frauds which are difficult both to foresee and to avoid; the owners seldom recognise the advantage to themselves of rooting out tuberculosis, which decimates their herds; the continual changes which are taking place amongst the stocks (sales

or exchanges of milch cows and fraudulent substitution) would necessitate the formation of a staff of inspectors and of numerous checks, whilst the astounding frequency of bovine tuberculosis in certain districts would practically destroy the milk trade if only milk from perfectly healthy animals were permitted to be sold.

For the time being, therefore, it seems impossible to make the tuberculin test obligatory for milch cows, but the practice should be strenuously encouraged as a private and voluntary means of protection.

In this connection the efforts made by certain towns like Nice and Cannes cannot be too highly praised. There the dairies are under inspection, the cows are regularly tested with tuberculin, and those establishments which place themselves under administrative control receive certain advantages. The municipal bodies in other towns might with great advantage organise a similar service.

Sanitary inspection of byres.—By periodically examining the byres it would be easy to detect all animals suffering from tuberculosis. Although milk from animals which only show trifling tuberculous lesions, inappreciable on simple clinical examination, may be virulent, yet as a general rule it contains too few bacilli to be really dangerous. The milk of cows which have fallen into poor condition as a result of generalised tuberculosis is often infected, and that of cows suffering from tuberculosis of the udder is rich in tubercle bacilli and very dangerous.

Generalised sanitary inspection of byres would result in cows suffering from any clinical form of tuberculosis being got rid of, and would thus ensure a relative degree of security to the public. The experiments carried out at Manchester by Delépine showed the real importance of simple clinical examination of the mammary glands and of the annexed lymphatic glands.

The sanitary control of the milk trade is of the greatest importance, a fact recognised by the last International Congress of Hygiene held at Brussels in 1903. M. Strauss emphasised its value by including in his proposed law for the protection of mothers and infants (the first reading of which has already been accepted by the Senate) a clause for the introduction into each department of a system of dairy inspection. It is to be feared, however, that the organisation of such a service over a large extent of country would prove difficult, and that numbers of small dairies would escape control. Although this may be possible, it need not be regarded as very serious. In point of fact, as Bang has shown, tuberculosis is much commoner in the larger establishments. Thus, of 122 dairies, each containing more than 50 cows, not one was found free from tuberculosis, whilst of 924 dairies, each containing less than 10 animals, 418 were completely exempt. The percentage of cases of tuberculosis in dairies rises in direct proportion to the number of animals in each. Of the 122 infected dairies, each containing more than 50 animals, 71, or 58·19 per cent., contained from 50 to 100 per cent. of tuberculous animals. Among 500 dairies, containing less than 10 animals each, only 164, that is to say, 32·34 per cent., contained as high a proportion of tuberculous patients. Similarly, of the 122 dairies included in the first category only 21, that is to say, 17·21 per cent., contained less than 25 per cent. of tuberculous animals, and of the 507 dairies included in the second category, 232, that is to say, 45·75 per cent., contained less than 25 per cent. of diseased animals.

The recent observations of Conte in ¦Hérault prove that in France the conditions are similar to those in Denmark. The chances of a byre becoming infected increase with the frequent introduction of fresh stock, and it is known that animals are most frequently changed in the largest establishments.

As a consequence, it is these which require the most stringent sanitary regulation, but the inspection even of small dairies could be carried out

without much expense by handing it over to the veterinary surgeons charged with the control of contagious diseases. France, which possesses about 8,000,000 milch cows, produces annually about 79,000,000 hectolitres of milk. Although this milk constitutes so large a proportion of human food and represents a money value of 1,200,000,000 francs, it is not at present subject to any systematic examination. The authors therefore consider it desirable that the production and sale of milk should as early as possible be placed under proper sanitary control.

Furthermore, it is desirable that public bodies should favour the practice of pasteurising milk and testing milch cows with tuberculin, by bringing to the notice of the public the dangers of consuming raw milk, and by deciding, in accordance with M. Strauss' proposition, that only pasteurised milk or milk from cows tested with tuberculin should be purchased for all public institutions like hospitals and the dispensaries of the various asylums, in which uncooked milk is too frequently consumed.

Finally, in anticipation of the inspection of dairies and the regulation of the milk trade, it is necessary to institute in the veterinary colleges classes for the inspection of milk, as is now done in the German veterinary colleges in conformity with the law of 1901.

The measures suggested may to some persons seem a little exaggerated, but they are not disproportionate to the gravity of the evil against which they are directed, as the following observations prove.

The tubercle bacillus occurs very frequently in milk sold in large towns. Since 1884 H. Martin has regularly examined samples of milk bought in Paris, and has found tubercle bacilli in 33 per cent. of the samples. At Naples 15 per cent., at Manchester 11 to 18 per cent., and at Berlin 30 per cent. of samples of milk offered for sale were found to contain tubercle bacilli.

Even although in a mixture of infected and normal milk the bacilli are sometimes sufficiently diluted to render them inocuous (a fact which led Gebhart to regard commercial milk as not very dangerous), this affords no security whatever under other circumstances. Tuberculous milk even after dilution with fifty times its original volume of liquid may retain its full power of infecting the consumer.

Substances prepared from milk, such as butter and cheese, may be regarded as practically non-injurious. Butter not infrequently contains bacilli. German investigators have found true tubercle bacilli in 7, 15, 29, 32, and even in 70 per cent., of the samples examined. As shown by Galtier, cheese may also contain virulent bacilli, but butter and milk always contain fewer bacilli than the milk from which they have been prepared, and are swallowed in such small proportions as to prove of little danger in practice.

On the other hand, the extreme frequency of tuberculosis in Danish and German pigs fed on skim milk shows how dangerous this bye-product is when prepared from milk containing tubercle bacilli. Such skim milk should be boiled or pasteurised when used for feeding either adults or infants.

Finally, although goats rarely suffer from tuberculosis, they should be included under the regulations prescribed for cows, for the milk of these animals, which sometimes show true tuberculous mammitis, is somewhat largely consumed by human beings.

Summing up the foregoing, the writers suggest the following draft regulations for acceptance :—

1. A system of sanitary inspection of dairies and regulation of milk supply should be instituted in each Department.

2. The system should be organised by the General Municipal Council, and be administered by the veterinary staff which usually deals with contagious diseases.

3. Any person wishing to sell milk should first be required to make a declaration to the Mayor of his Commune.

4. The dairies or cowsheds where the milk is produced should be thrown open for inspection.

5. These establishments should be inspected at least every two months, the points to be observed being the health of the animals and the hygienic conditions of the cow sheds.

6. The owners of animals yielding milk for public sale should be required immediately to report to the Sanitary Inspector any lesion of the udder which appears during the interval between two inspections.

7. Any animals which show clear signs either of abdominal or thoracic tuberculosis, or of tuberculosis of the udder, should be slaughtered.

8. No milk should be offered for sale from any animal showing any morbid change whatever of the udder. This milk after boiling could be used for feeding animals.

9. Dairy owners should be prohibited from offering for sale milk from cows suffering from diseases likely to render it injurious. These diseases would be defined by " Orders " of the Board of Agriculture.

10. The Inspectors should be at liberty at any time to take samples of milk from the shops or from the dairies themselves.

11. To cover the cost of inspecting the dairies and milk, the Local Authorities should be authorised to collect a tax not exceeding one franc per head for all cows submitted to inspection.

12. Any infraction of the clauses of the above law should expose the offender to a fine of five to fifteen francs. In case of repetition the offender might be imprisoned for a period not exceeding five days.

It is proposed to supplement the action of the above law by distributing posters or notices drawing attention to the danger of consuming raw milk, and particularly of feeding children with unboiled milk.

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A permanent committee for the prevention of tuberculosis has passed the following resolutions :—

Firstly.—That in public institutions of all kinds only pasteurised, boiled, or sterilised milk, or raw milk obtained from cows which have been tested with tuberculin and periodically subjected to inspection, should be consumed.

Secondly.—That a course of instruction in the inspection of milk should at once be instituted in the national veterinary schools. (Vallée and Villejean, *Rev. Gén. de Méd. Vét.*, No. 46, 1904, page 521).

AN OUTBREAK OF ANTHRAX IN HORSES.

ANTHRAX in horses is extremely rare in France. In the army a few cases have been reported from time to time, but not since 1872 has any considerable outbreak occurred. On that date a large number of horses were attacked at Tarascon. In 1882 sixty-six horses, constituting nearly half the equipment of a regiment, died of the disease at Montauban.

The outbreak reported by M. Bourgès occurred last year in the camp of Chambaran, in Dauphin, where 510 horses had been in camp since 19th July. A month later—*i.e*, on the 19th August—three cases of anthrax occurred, at intervals of a few hours, after the animals had returned from morning manœuvres, which had been carried out in heavy, cold rain.

The first horse was attacked at 10 A.M., and died at 4 P.M. The second showed signs of severe illness at 1.30 P.M., when it was being removed to the

infirmary. The third was seized at 2 P.M. It showed a slight swelling in the left parotid region. Death occurred twenty-seven hours later.

On examining these animals after death anthrax lesions were found. On the 17th of August twelve cast horses had been sold, and on the day before the appearance of the above three cases one of them showed signs of purpura hæmorrhagica; it died a week later. This case of spontaneous purpura somewhat obscured the diagnosis, for it would have seemed hasty to declare the existence of anthrax in a camp where a case had not been seen for twenty years. The uncertainty, however, did not continue for long.

On the 21st nine new cases, eight of which were extremely acute, removed any remaining doubt as to the nature of the disease, and, although microscopical examinations were not then made, the clinical appearances and the lesions found on *post-mortem* examination were sufficient on which to form a diagnosis, which was afterwards confirmed by the discovery of anthrax bacilli.

Causation.—Anthrax is regarded as almost invariably of alimentary origin. At Chambaran the forage was not beyond reproach. Before the appearance of the disease several samples of hay had been condemned. Some of it was very mixed, and contained various thorny plants, which might have served to prepare the way for later infection.

The oats were obtained locally, and were of good quality. The straw used for litter was often of second quality, and it seems possible that it may have contained germs of anthrax; some of the horses certainly ate this straw. If, however, one admits this method of contagion, it is difficult to explain why the cattle and horses on the farms from which it was obtained were not also infected.

The municipal veterinary surgeon of the district, who was in a position to form an opinion, refused to believe in the existence of anthrax in the camp, stating that the disease had been unknown in that district for many years. The sudden occurrence of anthrax in a dozen horses scattered throughout five stables, coinciding with torrential rains during the preceding days, raised a suspicion that the disease might have resulted from the drinking water becoming polluted by infiltration from the sides of a ravine through which the principal stream supplying water for the horses ran.

This ravine received drainage from the kitchens and from a little swampy pond, the water from which was more or less filthy and smelt offensively. The infiltration of this drainage, however, was not sufficient to prove contamination from that source. Moreover, no bacteriological examination of the water was made.

It is known that Koch has emitted views regarding anthrax which are quite opposed to those of Pasteur. He states that the bacillus only becomes parasitic by accident, and that it lives and multiplies as a saprophyte in water and in certain soils. This view is based on experimental and clinical facts which give it a certain plausibility, without, however, affording direct proof of its truth. We know, for instance, that the bacillus thrives well in decoctions of hay, grains, and roots, provided they are neutral or alkaline in reaction. Furthermore, it is known that anthrax is common in moist places, near ponds and small streams, and after floods.

Koch believes that the bacillus grows in stagnant water and on the surface of the ground or on plants, and forms spores during hot weather. If the water rises the spores are carried broadcast over the fields. He thus interprets the well-known influence of floods and of the proximity to streams and ponds.

Bourgès regards Koch's theory as less hypothetical than is generally supposed, and adduces the following facts observed during the last Chinese expedition as strongly supporting it.

At Yang-Tsoun two batallions of artillery and a squadron of Chasseurs

d'Afrique were posted. All the horses received similar food, but a limited number had a separate water supply. During September 1901 anthrax appeared in a battery having a special water supply. Nine horses or mules were attacked and six died. The disease only affected horses drinking from the special water supply, and when the supply was changed anthrax ceased.

At Chambaran, however, changing the water certainly did not check the disease in a clearly marked way. The first three cases occurred on the 19th of August and the last on the 5th September.

From the 20th to the 25th of August the horses drank from running water and from neighbouring ponds. On the 26th they received only the regular town water. After appearing suddenly and attacking twelve horses, eleven of which died in three days, the disease suffered a very marked check, for between the 22nd and the 28th only eleven new cases occurred, eight of which died.

Although the horses of the 5th Artillery were decimated by the disease, seventeen young animals belonging to the 2nd Artillery, which were isolated, remained unaffected. The food was similar in all cases, except that the last-named animals received different straw litter. These young animals did not drink from the suspected source. It might be urged, however, that this proves nothing, because many horses of the 5th Regiment escaped. Nevertheless, infection through the medium of the water seems to Bourgès the probable cause.

The period of incubation of anthrax varies within considerable limits. The natural course is seldom as rapid as the experimental. Colin and Pasteur found that if sheep were fed with lucerne, watered with cultures containing spores, many of the sheep escaped, even after appearing visibly ill, notwithstanding the enormous number of spores ingested. A smaller number died with all the symptoms of spontaneous anthrax after a varying period, sometimes lasting as long as eight or ten days. The majority, however, showed the common acute type of disease, in which the period of incubation is believed to be very short.

In the absence of exact observations regarding the incubative period in the horse, Bourgès thinks that spores of anthrax taken with the drinking water might perhaps have remained in the digestive tract for several days or even a week, awaiting some favourable combination of circumstances before making their influence felt.

Investigations relating to incubation periods show that for most diseases an average period can be fixed, but that great variations occur, from which anthrax is probably not exempt.

The *virulence* of the disease also varies within wide limits in all animals, and Bourgès believes that a considerable number of horses at Chambaran suffered from the disease without showing serious disturbance. The disease appeared suddenly after the heavy cold rain which had fallen during the morning manœuvres. The chill thus inflicted diminished the bodily resistance of the animals, and caused the disease to make rapid progress in those horses which had received the largest quantity of bacilli, or which proved most susceptible to their action.

Possible contamination of the soil.—At Chambaran the soil is composed of a mixture of clay and chalk. The layer of clay rests on two beds of glacial drift pebbles which are usually found near the surface of tertiary (Pliocene) formations. The soil is impervious to moisture. Before a permanent camp was set up in 1883 the ground was covered with trees. The part occupied by the firing butts is swampy and shows scattered pools of water, notwithstanding the fact that it stands nearly 1700 feet above sea level. Although the neighbouring peasants had never heard of the existence of anthrax in this district, it may nevertheless have existed. An animal dies either suddenly or

after a short illness, and is buried without any *post-mortem* examination. Such cases are often attributed by the farmer to colic, congestion, etc., and are buried more or less deeply without any precaution, thus forming centres for anthrax infection.

One method of infection is often overlooked, namely, dust carried by the wind. Anthrax has more than once been introduced with the artificial manures now so largely used, and anthrax-infected dust carried by the violent winds which sweep over the camp during the middle of August might possibly have formed a method of contagion. Such dust might fall into and infect the water supply. Bourgès considered that, although not all of equal importance, these points deserved consideration.

The path by which the virus enters the system. — Anthrax bacilli seem principally if not exclusively to obtain entrance into the body through the digestive tract. The excoriations, cracks, and injuries of the skin which are always common in horses employed on manœuvres cannot be blamed in this case. A horse with a slight wound on its knee died of anthrax, but the wound showed no sign of swelling at any time during the course of the disease, for which reason Bourgès did not regard it as the seat of inoculation. Another horse, fifteen years old, slaughtered on account of chronic nasal gleet and caries, showed during life some symptoms of anthrax which were confirmed by *post-mortem* examination. It seems just possible that this old-standing lesion in the sinus may have served as a point of entrance for the bacteria.

Four animals showed external swellings, but Bourgès does not think these indicated the site of infection. He regards the swellings merely as external manifestations of infection, which was already far advanced when the swellings were observed.

Course of the Outbreak.—On the first, third, and fourth days ten horses died. On the fifth day one, on the sixth two, on the seventh one, on the eighth two, on the tenth one, on the eleventh one, and on the thirteenth day (which was the 31st of August) one.

The disease then seemed to have ceased, and, as the remaining horses were in very good condition, they were returned to work on the twelfth day. On the fifteenth day four horses died. This seemed to show that return to work diminished the animals' power of resistance and thus favoured the action of the anthrax bacilli. Whatever the reason, the animals were withdrawn from work, and for a time were only exercised in hand. This precaution, and the removal of the horses to a distance of 4000 yards from the infected stables, was carried out on the sixteenth day, and was soon afterwards followed by the complete cessation of the outbreak, only one animal dying on the sixteenth, two on the seventeenth, and one on the eighteenth day.

A careful study of the positions occupied by the animals which died failed to reveal any evidence of contagion from one horse to the other.

Symptoms.—Anthrax commences with loss of appetite and slight colic. When these symptoms are sufficiently marked to attract attention the animals are already profoundly affected. Notwithstanding this, they do not on casual examination appear to be seriously ill, and were it not for the experience previously gained one would not recognise as diseased horses which are within a few hours of death (this, of course, applies only to superficial examination). The coat is not always staring, the animals move as usual, except that they are a little dull. The limbs are more or less cold, as are also some portion of the body, and certain animals may exhibit muscular trembling, *i.e.*, rigor. In rare cases the first patients seen may move stiffly and exhibit a staring coat, but, as a rule, they seem in health. When in this stage of the disease four of the twenty-seven horses which died showed specific swellings. In three cases these swellings rapidly spread over con-

siderable areas. In the first the left parotid region was swollen. In the second the swelling was in the region of the girth, in the third in the subglossal lymphatic glands, and in the fourth behind the mass of the lower lip.

Careful examination of the patients revealed very grave symptoms. The temperature was rarely less than 40°-41° C., the conjunctiva was infiltrated, often injected; in two cases it exhibited an earthy tint, and in two others petechiæ. The pulse was feeble but rapid, 80 per minute, the artery small and hard. At this time blood was withdrawn; 'it was of a dark-red colour. and escaped with some force from the jugular vein. Received into a tall glass, it formed a dark-red clot capped by a small yellow clot. The respiration was little disturbed, the rythm of the movements being normal in cases where no swelling had occurred around the trachea or bronchi. The mouth was dry; the animals attempted to eat hay and straw but refused oats; they sometimes drank with avidity. In some cases urine was abundant and limpid, but was usually scanty. In two cases it was blood-stained. The presence of blood upon the litter at once attracted attention. The diarrhœa mentioned by some writers was not present in these cases, the fæces sometimes being soft but usually retaining their normal appearance.

Graver symptoms follow with varying rapidity. In very acute cases the pulse becomes thready, and cannot be counted; the respirations are accelerated, the nostrils dilated. In one case Bourgès was struck by the violence of the heart beats, which could be distinctly heard. There is now stiffness about the loins and difficulty in movement, dull continued colic sets in, the animal lies down on its side, rises again, and goes through the same process until worn out, when it falls heavily, and soon dies. The agony, which is usually short, is generally preceded by cold sweats, rapid falling of the bodily temperature, and sometimes by very loud groaning.

Four horses which showed swelling about the trachea and bronchi, or œdema of the pharyngo-laryngeal region, extending over the head and neck, displayed intense roaring, and a discharge of frothy, yellowish serosity from the nostrils, which were widely dilated. Death from asphyxia occurred early.

Attempts to bleed from the jugular, for the purpose of obtaining blood for microscopical examination, failed. At this period of the disease blood withdrawn into a tall glass formed a uniformly dark-reddish or blackish diffluent clot.

Bourgès always failed to discover bacilli before the animal's death, though a drop of blood from the spleen when stained with fuchsin showed the specific organisms in large numbers.

In ten cases the disease proved fatal in a few hours. In the other seventeen death occurred from the first to the fourth day.

The ages of the animals varied from six to sixteen years.

Lesions.—The carcases were usually opened a few hours after death. They did not appear greatly distended with gas, and the natural orifices did not discharge any liquid, except in the cases of animals which had died of asphyxia due to swelling about the region of the neck; these showed a sero-sanguinolent discharge from the nostrils.

The subcutaneous connective-tissue exhibited hæmorrhagic spots and infiltration with yellowish serosity. In the swollen areas the interstices of the connective-tissue were filled with a sero-gelatinous material in the centre of which appeared a hæmorrhagic stain.

The abdominal cavity contained from 2 to 6 quarts of reddish serosity, the peritoneum was hyperæmic, and some of its folds were covered with prominent distended blood vessels.

The vessels of the large intestine (colon and cæcum) were greatly distended, and the attached double chain of lymphatic glands exhibited numerous hæmorrhagic centres surrounded by the same amber-coloured gelatinous

serosity. The blood vessels and lymphatic glands formed bulky masses of a brownish or deep yellowish colour, depending on the predominance of the liquid filling or surrounding them. This lesion is absolutely characteristic. Bourgès states that, despite his having carried out numerous *post-mortem* examinations on animals suffering from all kinds of diseases, he has never before seen it. The engorgement of blood vessels and the extravasation of serum or liquid or gelatinous serosity are always present.

The surface of the intestine, particularly of the large colon and cæcum, exhibits hæmorrhagic patches of varying size. In one of the eighteen horses which died the point of the cæcum was blackish in colour, and greatly thickened in consequence of extravasation of blood into its walls. In another case the third portion of the colon was greatly congested, blackish, and hæmorrhagic.

The stomach, small intestine, and floating colon seemed free from such lesions; only in one case did the small intestine appear markedly hyperæmic.

The large intestine and cæcum contained numerous oxyurides, sclerostomata, and some ascarides. The mucous membrane of these bowels exhibited reddish spots produced by the parasites, a great number of which, particularly of the sclerostomata, adhered firmly to the interior. These appearances were particularly noticeable in the first three horses which died, but also occurred, though in a less degree, in some of the other animals.

The wounds produced by certain of these worms may very possibly have favoured the absorption of bacilli, and so contributed to the death of several of the horses.

In most cases the liver was congested and friable; in some one of the lobes appeared discoloured, whitish, and as though parboiled. In acute cases, however, the liver often appeared normal.

The spleen was hypertrophied, swollen, and engorged with fluid blood. Its parenchyma was soft and brownish, and the whole organ sometimes weighed as much as eight pounds. In one case, in which the horse had shown hematuria throughout the course of the disease, the spleen appeared almost bloodless and of a salmon pink tint.

The kidneys were congested and surrounded by gelatinous amber-coloured serosity. The pancreas was often hyperæmic. The bladder contained reddish urine, in two cases closely resembling blood.

On opening the thoracic cavity the lungs were found collapsed and of a reddish colour, but when an animal had died from asphyxia, due to extensive swelling about the throat, the lung was largely engorged with black blood, the bronchi and trachea were filled with yellowish frothy mucus streaked with blood, and a similar fluid escaped from the nostrils.

The pleura and pericardium were more or less ecchymosed, and covered with rose- or amber-coloured exudate.

The heart was sometimes contracted, sometimes relaxed. The endocardium showed a few ecchymoses. The blood which it contained was usually very dark in colour, coagulated imperfectly, and soon broke down into a soft, blackish, semi-fluid jelly. This colour of the blood, however, is not characteristic; in some cases it appears red.

The myrocardium often appeared as though parboiled. When death had been rapid the muscle was of a deep-red colour. The base of the heart was frequently surrounded by a hæmorrhagic exudate overlaid with amber-coloured gelatinous serosity.

When the swellings about the throat were incised, one found a hæmorrhagic exudate surrounding the lymphatic glands, and an abundant yellowish jelly-like substance distributed over the pharynx and larynx, along the jugular furrow, and over the lower margin and lateral surfaces of the neck as far down as the cariniform cartilage.

The whole of the head sometimes appeared swollen when the œdematous swelling had originated in the sub-glossal lymphatic glands.

In animals which had resisted for two or three days the muscular tissue was usually of a characteristic salmon tint. In acute cases, on the other hand, it appeared of a light or dark-red colour.

Blood from the spleen was often examined, and all the preparations were found to contain numerous bacteria.

Treatment.—None of the drugs tested appeared to have any real effect, and Bourges recommends slaughtering the animals as soon as the diagnosis is made, and attempting by suitable measures to cut short the course of the disease.

An entire change in the feeding and water supply was made. On 3rd December the horses were moved four kilometres from the camp, and were picketed in the open. On the same day an acute case of anthrax occurred. On the 4th September another horse, seen to be ill at 2 A.M., died at 8 P.M. Finally, on the 5th September a third horse was seen to be ill, and died in a few hours. This was the last fatality. (Bourgès. *Recueil de Méd. Vét.*, 1904, p. 607.)

EIGHTH INTERNATIONAL CONGRESS OF
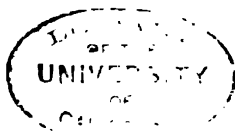# VETERINARY MEDICINE, BUDAPEST, 1905.

THE Organising Committee of the Eighth International Congress of Veterinary Medicine, which will be held in Budapest in 1905, has definitely decided on the programme of the questions to be dealt with at this scientific assembly. Eight questions will be debated in the Section of Veterinary Police, six in the Section of Physiology and Hygiene, and twelve in the Section of Pathology. Eighty distinguished specialists have already promised to present reports on these subjects. The questions of Veterinary Police will be discussed at meetings of the whole assembly, those of Physiology and Pathology at meetings of their respective sections. In the Reports and in the discussions, following the custom established in preceding Congresses, French, German, and English, as well as the Hungarian language, will be used. The original text of the memoirs, with an extract in several languages, will be sent in advance to members of the Congress, in order that they may acquire a detailed knowledge of the subjects to be dealt with.

To cover the expenses of the Congress, the Municipality of Budapest as well as the Ministry of Agriculture have promised sums in keeping with the importance of the Congress. Moreover, the General Assembly of the Society of Hungarian Veterinary Surgeons has voted a considerable allocation. The Society of Agriculture of Hungary have also promised their material help.

The invitations to the Foreign Governments will be issued by the Minister for Foreign Affairs at the request of the Ministry of Agriculture. Moreover, in order to increase the general interest in the Congress, and to assure a large number of adherents, the Committee have asked several eminent specialists abroad to form local committees in their countries. M. Barrier (Alfort), for France; Prof. Schmaltz, (Berlin) for Prussia; Prof. Ellenberger, for Saxony; Dr Lorenz, for Hesse; Prof. Perroncito (Turin), for Italy; and M. Degive (Brussels), for Belgium, have already replied that they are willing to undertake this task.

The Congress will be held from the 3rd to the 9th September, and the invitations to it will be issued shortly.

All letters and inquiries concerning the Congress should be addressed to Prof. Etienne de Rátz, Secretary for the Congress, Budapest, VII Rotten-biller-utcza 23.